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MIXED MESODERMAL TUMOURS OF THE UTERUS IN THE BANTU

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These tumours are sufficiently rare to be worth recording—a case has previously been described in a Bantu woman by Charlewood and Murray.[1] Since Wagner [2] described the first case of botryoid sarcoma in 1854, some 99 cases in all have been described (Charlewood and Murray [1]).

CASE 1

A Bantu female, aged about 48 years, was first admitted to King Edward VIII Hospital on 11 May 1949, complaining of a vaginal growth of 12 months' duration. In recent months she had noticed a thin watery vaginal discharge, occasionally blood-stained. She had had 13 full-term normal deliveries. Her menses had always been regular, lasting 4 days every month. Her last menstrual cycle was in March 1949.

Vaginal examination revealed a large grape-like cervical polyp arising from the posterior lip of the cervix. The polyp was removed by diathermy and submitted for histological examination. It was reported as an 'acutely inflamed cervical polyp'.

The patient was readmitted on 11 January 1951, with a similar history and clinical findings. A polyp was again removed with diathermy and sent for section. It was reported as an: 'ulcerated lobulated cervical polyp with no evidence of malignancy'.

The patient was next admitted to hospital on 6 June 1951 with a further recurrence of a blood-stained vaginal discharge. Vaginal examination revealed two soft ulcerated cervical polypi, arising from the posterior cervical lip. The uterus was mobile, normal in size and the fornices clear.

In view of the patient's age and the recurrent nature of the lesion, a total hysterectomy and bilateral salpingo-oophorectomy was performed on 12 June 1951. Hysterectomy was immediately preceded by vaginal amputation of the polypi. The parametrium showed no evidence of infiltration.

The pathological report on the specimens read as follows:

'*Sarcoma Botryoides*. The uterus and cervical polypi were received separately. The polypi are covered by squamous epithelium and show a myxo-sarcomatous stroma with bundles of spindle cells, and in one area multinucleated tumour giant cells. Mitoses are present but not numerous. Groups of elongated cells with eosinophilic cytoplasm resembling striated muscle fibres are present. The cervix shows smaller polypi with a similar structure. There is no evidence of extension beyond the cervix.

'The previous biopsies have been reviewed—the original polyp had a very cellular stroma but could not be classed as malignant. The second biopsy does reveal groups of striated muscle fibres, but the sarcomatous nature of the stroma is largely masked by infiltration of inflammatory cells.'

Fig. 1. Section of tumour showing striated muscle fibres (case 1). X1,000 H.E.

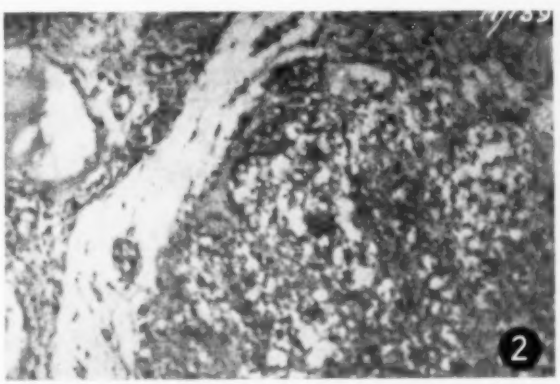

Fig. 2. Section showing embryonic sarcomatous stroma (case 1). X100 H.E.

The patient made a good immediate post-operative recovery, and no further treatment was instituted.

On 9 December 1952 the patient was again admitted. She complained of a vaginal growth of one week's duration associated with a yellow offensive discharge. The vaginal examination revealed a soft smooth elongated polyp arising high up from the anterior vaginal wall. The pelvis was free from infiltration. The patient's general condition was excellent. Radiographs showed no evidence of secondaries. The haemoglobin was 13.65 gm. %.

The polyp was removed at its base with diathermy and submitted for histological examination. The pathology report read:

'Vagina—Sarcoma Botryoides. Section of the polyp show a sarcomatous stroma with cartilage and primitive straited muscle fibres.'

Surgical removal was followed by a course of deep X-ray therapy.

CASE 2

A Bantu female aged 61 years was admitted to Eshowe Hospital in September 1951. She complained of a haemorrhagic vaginal discharge of 12 months' duration.

Vaginal examination revealed an ulcerated tumour protruding through the cervical canal.

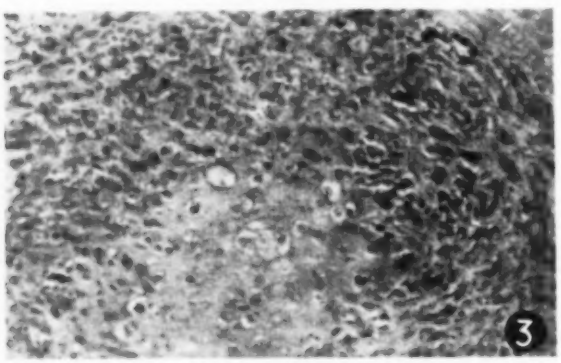

Fig. 3. Section showing sarcomatous stroma with cartilage formation (case 2). X100 H.E.

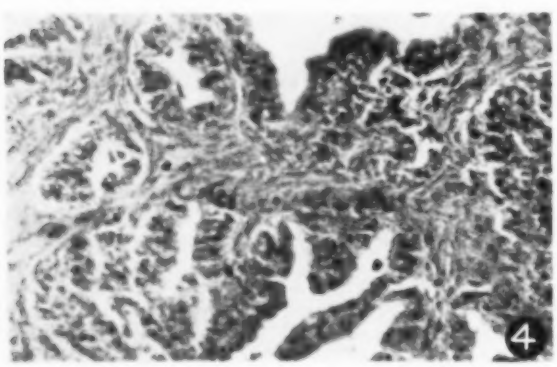

Fig. 4. Section of tumour showing adenocarcinoma (case 2). X100 H.E.

This was removed and sent for section. The pathological report on the specimen read as follows:

'Mixed mesenchymal tumour of the uterus. The ulcerated growth showed sarcomatous areas, large eosinophilic primitive muscle cells, areas of cartilage and undifferentiated mesenchyme. A very interesting feature is the carcinomatous change in the endometrial glands.'

The patient refused to have a hysterectomy done and was discharged from hospital. To date she had not again returned to hospital.

PROGNOSIS

The course is almost invariably fatal. The average duration of life after treatment is under one year (Novak [3]). Von Franque [4] had one 10-year cure.

Local recurrences and invasion of the pelvic organs and peritoneum are the usual sequelae. Metastases to the lungs and bones may occur.

TREATMENT

Total resection of uterus and adnexa is advisable. This should be followed by deep X-ray therapy (Charlewood and Murray [1]).

DISCUSSION

Mixed mesodermal tumours of the uterus fall into two main types:

1. Pedunculated cervical tumours occurring in young adults, the mean age being about 31 years (Willis [5]).

2. Corporeal growths which usually have a broad base in the fundus, and are usually post-menopausal.

As Pfannenstiel [2] (1892) earlier described, most of the cervical mixed tumours are conspicuously grape-like or botryoid, in this respect resembling the vaginal sarcomas of infants.

The corporeal tumours are bulky but this may be mainly due to mechanical factors.

They usually have a cellular sarcomatous stroma, often closely resembling embryonic mesenchyme. Cartilage (adult or embryonic) occurs in about three-quarters of the cases. Striated muscle fibres have been seen in about half the reported tumours (Willis [5]). Novak [3] states that embryonic striped muscle is the more frequently seen. Epithelial tissues in these growths are usually regarded as only included endometrium or cervical glands, and not an intrinsic part of the tumour. In a limited number of cases there is great concomitant hyperplasia if not neoplasia of glandular tissue. Rarely the epithelium becomes cancerous (Willis [5]). Case 2 is an example of this.

SUMMARY

Two cases of mixed mesodermal tumours in the Bantu are described. I have been able to find only one case previously recorded in the Bantu in the literature available. The interesting feature in case 1 is the relatively slow growth of the tumour.

I express gratitude to Dr. J. Parker, Superintendent of King Edward VIII Hospital, for permission to publish case 1, and to Mr. Gilbey, F.R.C.S. (Edin.), M.R.C.O.G., Senior Consulting Obstetrician and Gynaecologist, King Edward VIII Hospital, for helpful criticism and advice

My grateful thanks to Dr. D. Procter (Eshowe Hospital) for permission to use his case (case 2).

I am indebted to Dr. J. Wainwright for the histology reports and valuable advice.

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THE ULTRA-VIOLET FLUORESCENCE OF THE TONGUE IN AFRICAN CHILDREN

BERNARD T. SQUIRES, D.M.

Bechuanaland Protectorate

The ultra-violet fluorescence phenomenon of the tongue was described a quarter of a century ago by Hymans van den Bergh [1]; more recently Tomaczewski [2] has studied it, both in health and in relation to certain diseases.

Van den Bergh described a 'more or less visible red fluorescence' of the tongue; he found that if the tongue were scraped, the surface layer, consisting of bacteria, food particles and epithelial débris, exhibited fluorescence, but that the removal was only partial, for even after vigorous scraping the surface of the tongue retained some fluorescence.

Tomaczewski confirmed these findings, and noted that when the tongue is observed through a magnifier, the fluorescence is seen to be composed of small discrete fluorescent points, which he believed to correspond to the filiform papillae. The fluorescence varied considerably in different individuals, the incidence being greatest in the age group 0-20 years, in which 63 out of 71 healthy subjects (89%) showed it. With increasing age, the incidence declined to 47% in the 81-100 age group. Tomaczewski gave no indication of the differences in distribution or intensity of the fluorescence, but contented himself with classifying his cases as positive if fluorescence were present even to a very slight extent, and negative if it were not. Hagerman and Hirschfeld,[3] employing as subjects a selected sample of out-patients attending a dermatological clinic, made an attempt at classification, and a distinction between normality and abnormality. Of 543 subjects, 155 (29%) showed fluorescence extending over the entire lingual dorsum, and in another 174 (32%) at least half the area was covered; in the remaining 212 (39%) the fluorescence covered less than half the area or was absent. They considered the last group to be abnormal.

This paper, which is a preliminary communication, describes the appearance in African children of school age, inhabitants of the Bechuanaland Protectorate. The children lived the usual primitive rural life characteristic of the Tswana; their dietary habits and annual food cycle have been fully described elsewhere.[4]

Method. The source of radiation was a British General Electric 'Osira' mercury vapour lamp, enclosed in a light-tight box, from which the radiation emerged through a window 3" x 4".

Subjects. 873 African children of both sexes and apparently healthy, within the age range 7-18 years, were examined. As there was no significant difference in the results between the sexes both are considered together. Opportunity also arose for the examination of 44 European school children within the same age range and from the same area, the results of which are given for comparison.

Comment. The findings described above have been broadly confirmed, but there are a few points of difference. The fluorescence varies from a light red, or occasionally yellow-red, usually confined to the sides and back of the lingual dorsum in the neighbourhood of the circumvallate papillae, to a brilliant red which covers the entire dorsum down to the tip.

The results were classified in a manner similar to that of Hagerman and Hirschfeld (*supra*). Class 1 comprised those who exhibited a fluorescence extending over more than half the area of the dorsum, class 2 those whose fluorescence extended over less than half the area, and class 3 those in whom fluorescence was absent. The findings for both sets of subjects are given in Table I.

TABLE I: INCIDENCE OF FLUORESCENCE IN AFRICANS AND EUROPEANS

	Percentage of cases (to nearest unit)		
	Class 1	*Class 2*	*Class 3*
African	9	40	51
European	16	44	40

It will be noted that the percentage of European children in class 1 is nearly double that of the African children, but owing to the small size of the European sample, this difference must not be too greatly stressed.

In both cases the proportion of negative findings, 51% and 40%, respectively, is much greater than those given by Tomaczewski for the same age range (11%). The proportions of the various classes cannot, unfortunately, be properly compared with the findings of Hagerman and Hirschfeld, as the former employed a selected population of cases, whereas the present series represent a random sampling.

Tomaczewski states that the fluorescence is due to the bacterial production of porphyrins; there is, however, no evidence as to the origin of these compounds, which might be synthesized by bacteria, or decomposition products of haemoglobin derived from food or from the host. The significance of the porphyrins is also obscure at present.

In an attempt to discover whether or not there is any

nutritional significance attached to the phenomenon, Hagerman and Hirschfeld (*supra*) divided their dermatological cases, without reference to tongue fluorescence, into 2 groups according to whether their skin lesions were or were not considered to be associated with vitamin B deficiency. On comparison of the groups, the proportion of cases with defective or absent tongue fluorescence according to their classification was found to be significantly greater in those diseases supposed to be associated with vitamin B deficiency than in those with other skin disorders. They also gave preparations of vitamin B complex orally or by injection to 196 of their cases who showed abnormal fluorescence; in 141 (77%) the abnormality disappeared, and fluorescence increased.

According to Costello and Luttenberger,[5] the absence of flourescence in the area anterior to the circumvallate papillae is suggestive of vitamin deficiency, but they adduce no evidence in support of the hypothesis.

Observations extending over a period of 8 months have been made upon 87 of the present series of African children; this period included a summer vacation, during which Tswana children tend to improve their nutritional condition. In the months immediately after the vacation there was an increasing tendency to a drift from class 1 to class 2, and from class 2 to class 3, but the numbers involved were not significant. In a separate series, however, of 38 African children who presented well marked clinical signs of malnutrition, 5 (13%) showed a very faint fluorescence, the rest being negative.

Finally, the macroscopic appearance of the tongue bears no relation to the incidence of fluorescence, for tongues which appear normal often exhibit no fluorescence. There seems to be, however, according to investigations now in progress, a broad correspondence between the incidence of fluorescence and the ratio between the number of filiform and fungiform papillae per unit area of the tongue surface (papillary ratio), as calculated by the tongue print method.[6, 7]

SUMMARY

1. The phenomenon of ultra-violet fluorescence of the tongue in 873 African and 44 European children of school age is described.

2. For classification, the subjects were divided into 3 classes, (i) those who exhibited fluorescence extending over more than half the surface of the dorsum, (ii) those showing fluorescence over less than half this area, (iii) those who showed no fluorescence.

3. Nine per cent of the Africans fell into class 1, 40% into class 2, and 51% into class 3 as compared with 16%, 44% and 40% respectively of the Europeans.

4. A series of 38 African children who exhibited well marked clinical signs of general malnutrition were investigated. Five (13%) showed a very faint fluorescence, the rest being negative.

5. The macroscopic appearance of the tongue bears no relation to the incidence of fluorescence, but such incidence corresponds broadly with the papillary ratio of the tongue.

I am indebted to the Director of Medical Services, Bechuanaland Protectorate, for permission to record these findings, and to Mr. M. D. Watson for assistance with the examinations.

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ABSTRACT

G. Otto (1953): *Treatment of Gastric Patients by Roter Tablets.* Ther. d. Gegenw., **3**, 108.

This article is a report on treatment of cases of peptic ulcer and gastritis at the General Hospital Heidberg, Hamburg, with Roter tablets, of which the formula is given by the manufacturers as: Mag. carb. 0.4 gm., bism. subnitr. 0.35 gm., sod. bicarb. 0.2 gm., cort. rhamn. frang. and rhizom. calam. aa. 0.025 gm.

The 50 cases selected for the test were all in-patient cases in which positive results had not been obtained by other medical treatment during a period of 1–3 weeks in hospital and the patients were still complaining of symptoms. Every case was radiologically examined at the beginning and end of treatment and by fractional test meal and cholecystography. In selected cases the gastroscope was used, and the duodenal juice examined for cel.-content and for lambliae and amoebae. All cases with intestinal parasites were excluded. Somogyi's serum diastase test was used when diagnosis was doubtful.

The cases examined were 17 of duodenal ulcer (with gastritis), 13 of gastric ulcer (with gastritis), 10 of chronic hyperplastic or hypertrophic gastritis or gastritis granularis (without ulcer), 1 of phlegmonous gastritis and 9 of simple or anacid gastritis or gastro-duodeno-enteritis.

The average length of treatment with Roter tablets was 3.9 weeks, and with previous treatment the average length of hospital treatment was 6.5 weeks. The results were as follows:

In duodenal and gastric ulcer, there was marked relief from pain after 2 or 3 days' treatment and in all cases the ulcer was healed (except one case, F., 58, who had a large, bleeding, penetrating callous ulcer of the lesser curve, with a 15 years' history of gastric troubles).

In chronic hyperplastic or hypertrophic gastritis radiological examination showed a marked improvement in 4 cases, but not complete healing. In one case polypous changes in the mucosa of the antrum were healed.

It was found that this method of treatment was not successful in cases of anacidity, but only in cases showing at least subacidity. The gastric analysis in fasting patients showed that no change in the titre of acidity was produced.

In some cases the treatment was continued for several months and in none of these cases did a relapse occur or any severe discomfort suggestive of a relapse. The period of observation in these cases was limited to 9 months. No harmful action of the drug was observed and no case of bismuthism or epitheluria. In only one case did the tablets 'disagree', and in this case they were discontinued.

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VAN DIE REDAKSIE

KLINIESE NAVORSING

Een van die gevolge van die organisering van hospitale en ander kliniese dienste in die Verenigde Koninkryk in 'n Nasionale Gesondheidsdiens (NGD), was om meer as ooit die noodsaaklikheid van nasionale koördinasie van kliniese navorsing aan die lig te bring. Origens het die bestaan van die NGD die geleentheid gebied om daardie koördinasie te bewerkstellig. Hierdie feite het gelei tot die stigting in 1951 van 'n Gesamentlike Komitee van die Mediese Navorsingsraad, wat op hierdie gebied vir meer as 30 jaar optree, en die staande Mediese Adviserende Komitee van die Ministerie van Gesondheid wat vir die NGD in Engeland en Wallis verantwoordelik is. Die Departement van Gesondheid vir Skotland onderskryf ook die bevindings van die Gesamentlike Komitee, wat in sy verslag *Clinical Research in Relation to the National Health Service** uiteengesit word.

Die uitdrukking ‚kliniese navorsing' word in die verslag gebruik om ‚nie slegs werk aan pasiënte in die hospitaal nie, maar ook veldstudies in epidemiologie en maatskaplike geneeskunde en waarnemings in algemene praktyk' te dek. Dit ‚strek van waarnemings wat bykomstig is tot en onafskeidbaar is van goeie praktyk, tot sistematiese ondersoeke wat opsetlik en oor lang periodes onderneem word, met die oogmerk om spesifieke vrae te beantwoord'. Sodanige navorsing is in die laaste jare met toenemende intensiteit uitgevoer, en is 'n stoot vorentoe gegee deur die aanstelling deur universiteite van voltydse kliniese professore en navorsingswerkers en die skepping van professorale eenhede, en deur die navorsingseenhede van die MNR en privaat navorsingsstigtings.

Die Gesamentlike Komitee het besef dat ‚die hele navorsingsstruktuur van 'n land, in die laaste instansie, op sy universiteite gebaseer is'; maar dat, weens verskeie redes, die universiteite nie in staat is om die hele gebied van kliniese navorsing te dek nie; en dit is by die aanvulling van die universiteite se bydrae waar die hoofrol van die Navorsingsraad lê. Sedert die stigting van die NGD is kliniese werkers en kliniese fasiliteite in Groot Brittanje vir praktiese doeleindes slegs binne daardie diens beskikbaar, dit is derhalwe die diens se plig om daardie werkers en fasiliteite vir navorsing beskikbaar te maak.

Die Gesamentlike Komitee se aanbevelings val onder drie hoofde:

1. *Sentrale Navorsingsorganisasie.* Dit word aanbeveel dat 'n Kliniese Navorsingsraad deur en as 'n Komitee van die MNR gestig moet word in ooreenstemming met die Ministerie van Gesondheid (England en Wallis) en die

* S.O. Code No. 45-12. London: H.M. Stationery Office.

EDITORIAL

CLINICAL RESEARCH

One of the results of the organization of hospitals and other clinical services in the United Kingdom into a National Health Service has been to make more apparent than ever the need for national co-ordination of clinical research. Moreover the existence of the NHS has afforded the opportunity of bringing that co-ordination about. These facts led to the setting up in 1951 of a Joint Committee of the Medical Research Council, which has been in operation in this field for more than 30 years, and the Standing Medical Advisory Committee of the Ministry of Health, which is responsible for the NHS in England and Wales. The Department of Health for Scotland also is in agreement with the findings of the Joint Committee, which are set out in its report *Clinical Research in Relation to the National Health Service.**

The term 'clinical research' is used in the report to cover 'not simply work on patients in hospitals, but also field studies in epidemiology and social medicine and observations in general practice'. It 'ranges from the making of observations which are incidental to and inseparable from, good practice, to systematic investigations undertaken deliberately and over long periods, with the object of answering specific questions'. Such research has been carried on of late years with increasing intensity, and has been forwarded by the appointment by universities of whole-time clinical professors and research workers and the creation of professorial units, and by the research units of the MRC and private research foundations.

The Joint Committee recognizes that 'in the last analysis the whole research structure of a country is based upon its universities'; but that for various reasons the universities are not able to exploit the whole field of clinical research; and it is in supplementing the universities' contribution that the primary rôle of a Research Council lies. Since the establishment of the NHS, clinical workers and clinical facilities in Great Britain are for practical purposes available only within that Service, on which therefore falls the duty of making those workers and facilities available for research.

The Joint Committee's recommendations fall under three headings:

1. *Central Research Organization.* It is recommended that a Clinical Research Board should be established by and as a committee of the MRC in agreement with the Ministry

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Departement van Gesondheid vir Skotland. Hierdie voorgestelde Raad moet verantwoordelik wees vir die bevordering van navorsing op 'n Verenigde Koninkryk basis, en hierdie funksie moet dit hoofsaaklik uitvoer deur (1) die toestaan van navorsingstoekennings om ‚gedesentraliseerde' navorsing deur plaaslike hospitaalowerhede en individuele werkers aan te help en (2) die samestelling van kliniese navorsingseenhede wat, hoewel hulle in die meeste gevalle by NGD hospitale, as deel van die plaaslike hospitaal personeel werk, onder beheer van die Raad sal bly.

Die Raad moet bestaan uit persone (11 is die voorgestelde getal) met ondervinding op kliniese- en navorsingsgebied, en die sekretaris daarvan moet 'n senior mediese beampte van die MNR wees. Sy fondse moet deur die MNR voorsien as die beherende liggaam voorsien word, en dit word aan die hand gedoen dat sy jaarlikse uitgawe in 3 of 4 jaar tyd op £250,000 te staan moet kom. Die MNR spandeer alreeds oor die £430,000 per jaar op alle afdelings van mediese navorsing, en dit word in die vooruitsig gestel dat die fondse wat nou vir kliniese navorsing voorgestel word, addisioneel moet wees.

2. *Gedesentraliseerde Navorsing Ondersteun met Skatkisfondse.* Dit is dan die voorneme dat, benewens navorsing wat direk onder die voorgestelde Kliniese Navorsingsraad georganiseer word, kliniese navorsingsprojekte van plaaslike hospitaalowerhede (Hospitaalstreekrade, Beheerrade, en Hospitaalbestuurkomitees) deur middel van die Kliniese Navorsingsraad voorsien uit skatkisfondse ondersteun moet word. Plaaslike hospitaalowerhede sal nog by magte wees om navorsing uit hulle eie fondse te finansieer. Dit word beskou dat ‚gedesentraliseerde' navorsing nodig is vir die ontdekking en aanmoediging van talent en om die lus vir navorsing by kliniese praktyk aan te moedig. Dit is die bedoeling dat dit die grootste moontlike mate van vryheid van gedetaileerde sentrale toesig sal geniet; maar dit word nogtans voorgestel dat goedgekeurde plaaslike navorsing gekoördineer moet word met die nasionale program van werk en dat sekere perke op individuele toekennings en die salarisse van voltydse navorsers in plaaslike skemas geplaas moet word.

3. *Loopbane in Kliniese Navorsing.* Die Gesamentlike Komitee dring aan dat dit belangrik is dat mediese praktisyns wat hulself aan kliniese navorsing wy, nie as gevolg daarvan enige professionele nadeel moet ly nie. Dit is waar dat dit in baie gevalle nie mense van navorsing weerhou het nie, maar nogtans ‚is dit onwaarskynlik dat enige skema vir loopbane in kliniese navorsing wat nie terme en voorwaardes netso aantreklik as die beste in geneeskunde voorsien nie, die vereiste kwota van bekwame manne in sy diens sal hou nie'. Dit word derhalwe voorgestel dat, beide in die junior en senior geledere, loopbane in kliniese navorsing in ooreenstemming gebring moet word met loopbane in die NGD, en dat die verwisselbaarheid so volmaak as moontlik moet wees; d.w.s. dit moet moontlik wees vir 'n kliniese beampte om met navorsing voort te gaan sonder nadeel tot sy professionele belange, en omgekeerd. Daar moet ook met die kliniese afdelings van universiteite verwisselbaarheid wees.

Die Gesamentlike Komitee lê ook nadruk daarop dat ‚kliniese navorsingswerkers dieselfde hospitaalstatus as ander kliniese beamptes moet hê. Daar sal algemeen mee saamgestem word dat kliniese navorsing nie afhanklik

of Health (England and Wales) and the Department of Health for Scotland. This proposed Board is to be responsible for the promotion of research organized on a United Kingdom basis and this function it should fulfil mainly by (1) the giving of research grants to facilitate 'decentralized' research by local hospital authorities and individual workers, and (2) by the setting up of clinical research units which, though in most cases working at NHS hospitals as part of the local hospital staff, will remain under the control of the Board.

The Board is to consist of persons (11 is the proposed number) experienced in the clinical and research fields, and its secretary would be a senior medical officer of the MRC. Its funds would be supplied by the MRC as its parent body, and it is suggested that its annual expenditure should amount to £250,000 in 3 or 4 years' time. Already the MRC is spending over £430,000 a year on all branches of medical research and it is contemplated that the funds now proposed for clinical research should be additional.

2. *Decentralized Research supported by Exchequer Funds.* The intention, then, is that besides research organized directly under the proposed Clinical Research Board, clinical research projects of local hospital authorities (Regional Hospital Boards, Boards of Governors, and Hospital Management Committees) should be supported by Exchequer funds provided through the Clinical Research Board. It will also still be competent for the local hospital authorities to finance research out of their own resources. It is considered that 'decentralized' research is necessary for the discovery and encouragement of talent and to foster the research spirit in clinical practice. It is intended that it shall have the greatest possible degree of freedom from detailed central supervision; but it is nevertheless proposed that approved local research should be co-ordinated with the national programme of work and certain limits placed on individual grants and the salaries of whole-time research workers in local schemes.

3. *Careers in Clinical Research.* The Joint Committee urges that it is important that medical practitioners who devote themselves to clinical research should not be under any professional disadvantage on that account. It is true that this has in many instances not held back people from research, but nevertheless 'any scheme for careers in clinical research which does not provide terms and conditions as attractive as the best in medicine is unlikely to maintain in its service the requisite quota of able men'. It is therefore proposed both in the junior and senior ranks that careers in clinical research should be equated to careers in the NHS, and that there should be as complete interchangeability as possible; that it to say, it should be possible for a clinical officer to move into research without detriment to his professional interests, and *vice-versa*. There should also be interchangeability with the clinical departments of universities.

The Joint Committee also urges that clinical research workers must have the same hospital status as other clinical officers. 'It will be generally agreed that clinical

kan wees van toegang tot pasiënte onder ander se sorg nie. Senior kliniese navorsingswerkers moet derhalwe die volle status van konsulterende artse of spesialiste hê'. Junior navorsingswerkers sal onder die senior navorsingswerker of klinikus in bevel werk, wat volle kliniese verantwoordelikheid vir die pasiënte het: hulle moet dieselfde status en kliniese verantwoordelikheid hê as werkers van vergelykbare senioriteit in die NGD. Dit word ook as belangrik beskou dat lede van navorsingseenhede die geleentheid moet hê om altyd in voeling met die algemene kliniese werk van die hospitale te bly.

research cannot be dependent on access to patients under the care of others. Senior clinical research workers must therefore have the full status of consultants or specialists'. Junior research workers will work under the senior research worker or clinician in charge who has full clinical responsibility for the patients; they must have the same status and clinical responsibility as workers of comparable seniority in the NHS. It is also considered important that members of research units should have facilities for continuing in touch with the ordinary clinical work of the hospitals.

SERUM PROTEIN-BOUND IODINE (PBI) IN THYROID DISEASE

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During the last decade considerable improvements have been made in the methods for the determination of the blood iodine, the protein-bound fraction of which appears to be identical with thyroid hormone. Although the technical difficulties are still considerable, the stage has been reached where these estimations can be carried out in a clinical laboratory. It may, therefore, be of interest to discuss the significance of the blood iodine and to record our results in 60 patients. An attempt has also been made to correlate these findings with the Basal Metabolic Rate and Radioactive Iodine Uptake where these studies were also carried out.

Baumann in 1895 first demonstrated the presence of iodine in the thyroid gland. The first estimations of blood iodine date back to 1899, but only the total blood iodine was estimated. Since the total iodine includes inorganic iodine as well as protein-bound iodine (PBI), the values were not always indicative of thyroid activity and were obviously susceptible to variations in diet, medication and probably other exogenous factors. With the unravelling of the complex biochemistry of the thyroid hormone, due largely to the brilliant series of researches initiated by Harington and his co-workers and summarized by Harington (1951) in his Richardson Lecture, considerable progress has been made in the development of micromethods for the estimation of the minute amounts of serum iodine and for the characterization of the protein-bound fraction. There is now good evidence that the thyroid hormone is thyroxine, which is normally stored in the thyroid follicle in protein combination as thyroglobulin. The circulating protein-bound iodine in the plasma or serum appears to be in large part thyroxine, although its exact chemical nature is as yet unknown. Taurog and Chaikoff (1946) have shown that in the normal animal the PBI is dependent upon the thyroxine content of the thyroid gland and is limited by the gland's capacity to produce thyroxine. Taurog and Chaikoff (1948) also found that 73-93% of the plasma PBI behaves like thyroxine in its solubility properties. Further, Peters and Man (1948) and Riggs (1947) conclude that the PBI may be taken as an index of the level of circulating thyroid hormone. Bassett, Coons and Salter (1941) showed that the PBI is present in the 'crude albumin' fraction of the serum.

More recent observations (Chesky *et al.*—1953) suggested that the determination of serum thyroxine gives a more reliable indication of the functional state of the thyroid gland than does determination of the PBI, and this modification of the serum PBI method may become the method of choice.

When radioactive iodine became available, a new era in the study of the physiology of the thyroid gland was opened. Hertz, Roberts and Evans (1938) initiated these studies in rabbits, and Hamilton and Soley (1939) in man, using the radio-iodine ^{128}I. These studies served to introduce the use of the radioactive isotopes of iodine as tracer substances in the study of thyroid physiology and disease. When the more suitable, longer-lived, isotopes ^{130}I and ^{131}I, particularly the latter, became available, more extensive clinical and physiological studies were feasible. The *in vivo* and *in vitro* studies of Chaikoff and Taurog (1949) and their collaborators have largely contributed towards elucidating the steps whereby inorganic iodides are converted to di-iodotyrosine, to thyroxine, and subsequently to thyroglobulin. It has been shown that in the biosynthesis of thyroxine in the thyroid gland, a complex enzyme system undertakes two oxidative processes. Firstly, iodide is oxidized to iodine, which then iodinates tyrosine to form di-iodotyrosine. Secondly, 2 molecules of di-iodotyrosine couple to form thyroxine. Raben and Astwood (1949) have reviewed the use of radio-iodine in studying the absorption, distribution and excretion of iodine in the body, and in throwing light on the methods by which iodine is bound into organic compounds and by which thyroid hormone is produced and discharged from

the gland. They have demonstrated its usefulness in studying the action of the anti-thyroid agents. It is not proposed to discuss here the developments in the use of radioactive iodine as a tool in the diagnosis of thyroid disease and in the many recent improvements in techniques. For these the reader is referred to the excellent reviews of Raben and Astwood (1949) and Cope (1952), and of Weinbren (1950), who also records his experience with radioactive iodine in South Africa. It is probably true to say, however, that the greatest value of radio-iodine has been in the study of thyroid physiology rather than in the diagnosis of thyroid disease.

ESTIMATION OF SERUM PROTEIN-BOUND IODINE

Since the concentration of PBI in the blood is very small and of the order of a few micrograms per 100 millilitres, reliable micromethods capable of detecting less than 0.1 μg. of iodine are required. With such methods it has become feasible to estimate PBI in 1 ml. of serum. The historical background to these attempts will not be discussed here and the reader is referred to the comprehensive review of Rapport and Curtis (1950). Until comparatively recently, however, the iodine was determined by one of the standard procedures, such as sodium thiosulphate titration. Sandell and Kolthoff (1937) introduced a new principle, viz. the microdetermination of iodine by its catalytic effect on the arsenious-acid reduction of yellow ceric ions to colourless cerous ions. The rate of decolorization of the ceric ions is followed in a photoelectric colorimeter or spectrophotometer, and the iodine concentration determined by comparing the rates of decolorization of test and standard iodine solutions. To determine the serum protein-bound iodine fraction, the serum protein is precipitated and the protein is either digested with concentrated acids or combusted in a furnace with the liberation of the iodine in the inorganic form. We first tried the former method. The modification we used was the digestion-distillation method of Connor *et al.* (1949). After precipitation of the proteins by the Somogyi zinc sulphate and sodium hydroxide reagents the proteins were digested by a mixture of chromic and sulphuric acids, following which the iodine was distilled over with phosphorous acid, and determined in the distillate by the catalytic reduction of ceric ions. We finally abandoned the method, since the results were extremely variable, this being apparently due to contamination of reagents. Van Zyl (1951) reporting his experience over several years states that the major disadvantages of the distillation technique are the ready contamination of solutions and the unsatisfactory methods for purifying reagents. He concludes that the reliability of the distillation method is unsatisfactory for accurate research purposes. Because of the technical difficulties involved in the distillation procedures, Barker *et al.* (1951) evolved a modification of the alkaline-incineration technique of Salter and McKay (1944) which appeared very suitable for a clinical laboratory. The serum proteins are precipitated with the Somogyi reagents, washed free of contaminating inorganic iodide, dried with alkali and incinerated at a temperature of 600°C ± 25°C in a muffle furnace to destroy protein and organic matter. The liberated iodine does not volatilize since it is retained by the alkaline ash. It is dissolved when cool in sulphuric acid and hydrochloric acid, after which it is determined colorimetrically by its catalytic effect as described above. We have found this method quite satisfactory when hydrochloric acid was omitted. This was necessary since we have not been able to obtain any batch of this reagent which did not give high blank readings. We are at a loss to explain our difficulty in this regard as all brands of HCl tested were found unsuitable. This applied to locally-manufactured HCl and that obtained from British and American chemical supply houses. Purification of HCl by distillation as suggested in a personal communication by Barker was tried without success. It appears to us that chloride, like iodide, catalyses the reduction of ceric ions by arsenious acid, and since Moran (1952) also states that the chloride ion interferes with iodine estimations, we tried Barker's method without HCl. Excellent blanks were now obtained. We found that the catalytic effect of concentrations of iodine similar to those of serum was slower than described by Barker *et al.* (1951) and was also somewhat depressed by the presence of sulphate ions. We therefore added sodium chloride to the arsenious acid reagent to improve the sensitivity. We determined by experiment the concentration of chloride ions that would approximately balance the effect of the sulphate ions, so that with a relatively small increase in the blank values, a concentration in the final test solution of 0.1 μg. of iodine (equivalent to 10 μg.

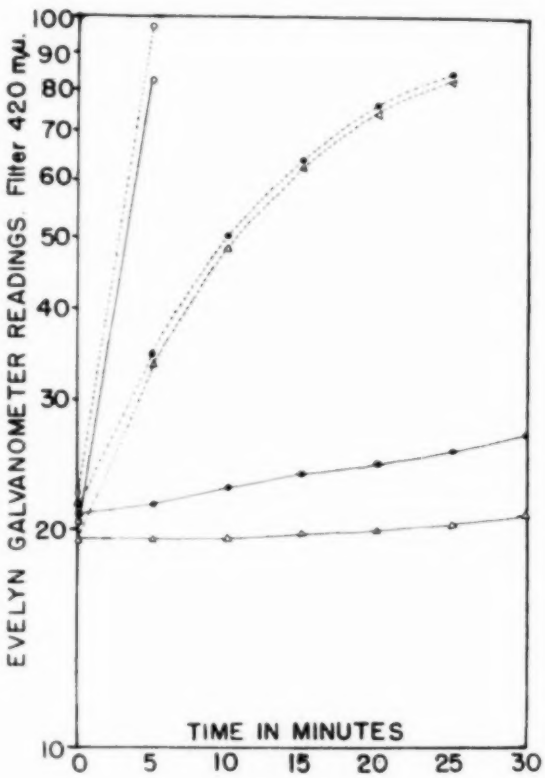

FIG. 1

Effect of Sulphate and Chloride Ions on the Iodine Catalysis of Arsenious Acid-Ceric Sulphate Reaction.

Reaction without Sulphate or Chloride.-
•—• without Iodine; •--• with 0·1 μg Iodine
Reaction with HCl:-
o—o without Iodine; o--o with 0·1 μg Iodine
Reaction with H_2SO_4 :-
Δ—Δ without Iodine; Δ--Δ with 0·1 μg Iodine

per 100 ml. of serum, since 1 ml. of serum is used for each estimation) produced an almost complete disappearance in the yellow colour of the final ceric solution after 30 minutes. An amount of 2 mg. of sodium chloride in the reaction mixture was found to be adequate for this purpose. We therefore added 0.2% sodium chloride in the arsenious acid reagent, since 1 ml. of reagent is used in each estimation. Although iodide is considerably more than 10,000 times as potent as chloride in catalysing the reduction of ceric ions by arsenious acid, we have not been able to confirm Barker's (1948) finding that the chloride ion only enhances iodide catalysis, and does not catalyse the reaction *per se*. Barker states that the addition of 1–3 mg. sodium chloride per tube made no appreciable difference to the blank values. Our figures however show proportionately increasing decolorization of blanks with increasing amounts of sodium chloride over the same range as used by Barker (1948). We are at a loss to explain this discrepancy and are engaged at present in investigating whether some reaction with the sulphuric acid used is producing 'non-specific' decolorization of the ceric solution.

Figure 1 shows the effect of sulphate and chloride ions on the iodine catalysis of the arsenious acid-ceric sulphate reaction. The influence of 2 ml. of 2N hydrochloric acid and of 2 ml. of 7N sulphuric acid on the rate of decolorization of 1 ml. of 0.02N ceric ammonium sulphate produced by 1 ml. of 0.1N sodium arsenite, all reagents made up as described by Barker (1951), is shown with and without the addition of 0.1 μg. of iodine (as potassium iodide) in a final reaction mixture made up to 10 ml. The slight depression produced by the sulphate ion on 'blank' solutions, i.e. containing no iodine, and the marked catalytic effect of the chloride ion are clearly shown. The latter is so great that the yellow colour of the ceric ion is almost completely discharged after 5 minutes (the '100' reading on the Evelyn galvanometer being equivalent to a completely colourless solution). After the addition of 0.1 μg. of iodine, the retarding effect of sulphate ion on the marked catalytic effect of the iodine is not significant, whereas in the presence of chloride the iodine effect cannot be reliably determined.

METHOD

The method we use at present differs from that of Barker *et al.* (1951) in the following details:

Reagents. Sodium arsenite, 0.1N reagent. This contains 0.2% sodium chloride for the reasons given above.

Iodine Standards. (a) Concentrated Stock Iodide Solution: (contains 100 μg. I/ml.). 130.8 mg. of desiccator-dried potassium iodide is dissolved in water and made up to 1 litre. The solution is kept in a brown glass bottle in the refrigerator. (b) Dilute Stock Iodide Solution: (contains 1 μg. I/ml). 1 ml. of the Concentrated Stock Iodine Solution is diluted to 100 ml. with water, and stored in a brown bottle in the refrigerator. (No change has been noted in these solutions after 6 months.) (c) Standard Iodine Solution: (contains 0.1 μg. I/ml.). This solution is made up fresh on the day of the test by diluting (b) 1:10.

Analytical Procedure. Each serum was analysed in duplicate. A series of specimens was analysed at one time.

1. *Precipitation and washing of plasma proteins*: as described by Barker *et al.* (1951).

2. *Drying and incineration*: Since at the altitude of Johannesburg water boils at 94° C, the tubes are placed in an oven set at 80–90° C to drive off water, instead of 85–95° C as described by Barker (1951). In other respects Barker's instructions were followed exactly.

3. *Dissolving iodide from the ash*: 2 ml. of 7N H_2SO_4 are added with due caution to avoid excessive effervescence. A glass rod is used to mix any unreacted ash with the acid and 8 ml. of water are added. The contents of the tube are stirred until the reaction appears to be finished and the tube is then centrifuged for 10–15 minutes at 2000 revs. per minute to pack the insoluble material.

4. *Determination of iodide*: Since, as mentioned above, the iodide catalytic effect is slower than described by Barker *et al.* (1951) each sample is not divided into two aliquots but the largest volume that can be pipetted off without disturbing the centrifugalized deposit (8 ml.) is taken for each estimation. This quantity is pipetted from each specimen into an Evelyn colorimeter tube, 1 ml. of the sodium arsenite solution is added to each tube, and the solutions mixed. The colorimeter tubes are then placed in a water-bath at 37° C for 10 minutes to come to temperature. The ceric solution is also warmed in the bath. Since the actual determination of iodine is accomplished by measuring iodide catalysis of the rate of decolorization of yellow ceric ammonium sulphate by arsenious acid, the times at which colorimeter readings are to be made must be determined beforehand. One ml. of the ceric solution is added at 1-minute intervals to each of the serum specimens and to a series of iodine standards which are run at the same time.

Iodine standards: 6 pyrex tubes as used for each serum specimen are taken, 8 ml. water, 1 ml. 10% zinc sulphate and 1 ml. 0.5N NaOH are added and these tubes are carried through steps (1) and (2) of the procedure exactly as for serum. In step (3) after additions of the 2 ml. of H_2SO_4, varying quantities of iodine standard (c) are added and the volume made to 10 ml. with distilled water. 8 ml. of each solution is then treated as in step (4). (For example, 0, 0.05, 0.10, 0.15, 0.20, 0.25 micrograms of iodide are added, corresponding to 0, 5, 10, 15, 20, 25 μg. per 100 ml.)

Colorimeter readings are made on the Evelyn photoelectric colorimeter using filter 420 and 10 ml. aperture at 0, 10, 20, 30 minutes. The results of each serum are read off from the standard curves prepared for each of these times. The particular curve used to read off each serum is selected on the basis of the time required to obtain a reading on the most sensitive part of the Evelyn colorimeter galvanometer (approximately 50 or mid-scale). Readings for normal individuals and hypothyroid patients are usually taken at 30 minutes. In hyperthyroidism 10-minute readings have been found most suitable.

RESULTS

The results obtained in 10 normal, apparently euthyroid, subjects are shown in Table I. The range 2.2–6.8 micrograms % is lower than the generally accepted range of approximately 4–8 micrograms %. (Rapport and Curtis—1950). This difference may be due to technical causes, or may possibly indicate some iodine deficiency in the young adult South Africans tested. This possibility is further elaborated below.

TABLE I: SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN TEN NORMAL SUBJECTS

Subject	*Sex*	*Age*	*Serum PBI μg. %*
1	M	39	5·4
			4·0
2	M	39	6·8
			5·3
3	F	29	5·6
4	F	28	5·8
5	F	35	4·7
6	F	25	3·4
7	F	33	2·6
8	F	19	4·1
9	F	21	2·2
			4·3
10	F	22	4·0

The results in our first 60 patients are recorded in Table II together with the salient clinical notes, basal metabolic rates and radio-iodine studies where these were also carried out. In figure 2 the results are classified according to the clinician's final decision. Though the hazards of clinical appraisal are well known in thyroid disease, the

TABLE II: SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN 60 PATIENTS

Case No.	Patient	Sex	Age	Case Notes	Final Clinical Assessment of Thyroid State	Serum PBI µg.%	B.M.R. %	Radioactive Iodine (^{131}I) Tracer Test				
								Dose	1 hour Uptake %	24 hours		
										Uptake %	Conversion Ratio %	Excretion %
1	M. N.	F	37	Slight nodular enlargement of thyroid. ? Anxiety state. Clinically no obvious evidence of thyrotoxicosis despite ^{131}I result.	Euthyroid	4·5	Normal	121 µc.	25·0	63·0	56·0	24·0
2	L. S.	M	—	Cardiac. On thiouracil for some months. Plasma cholesterol 204 mg.%.	Euthyroid	3·3	—	87 µc.	28·0	67·0	58·0	18·0
3	L. C.	F	41	Myxoedema. Thyroid gland not palpable. Improved on treatment with thyroid tablets.	Hypothyroid	0·6	Minus 40	—	—	—	—	—
4	V. W.	F	3/12	? Mongol. ? Cretin. Retarded bone age. Thyroid not palpable.	Hypothyroid	0·6	—	—	—	—	—	—
5	C. B.	M	40	Coronary atherosclerosis. Obesity. Thyroid normal in size. Plasma cholesterol 182 mg.%.	Euthyroid	5·2	—	—	—	—	—	—
6	A. V.	F	33	Thyrotoxicosis. Gross diffuse enlargement of thyroid gland. Thyroidectomy with clinical improvement.	Hyperthyroid	24·0	Plus 88	—	—	—	—	—
7	L. K.	F	37	Anxiety state. Thyroidectomy for non-toxic goitre producing pressure symptoms. Thyroid gland not palpable.	? Hypothyroid	0·6	—	—	—	30·6	21·0	68·4
8	D. S.	F	56	Angina of effort. Migraine. Xanthomatosis. Plasma cholesterol 740 mg.%.	Euthyroid	3·6	Minus 5	106 µc.	16·0	49·5	35·0	41·0
9	J. G.	M	44	Thyrotoxicosis. Slight uniform enlargement of thyroid gland. Treated with ^{131}I with excellent response.	Hyperthyroid	12·0	Plus 47	108 µc.	22·0	65·5	52·0	26·9
10	T. Z.	F	51	Toxic adenoma of thyroid. Removed surgically with complete clinical cure.	Hyperthyroid	10·1	—	—	—	—	—	—
11	H. J.	M	4½	Chondro - osteo - dystrophy. Thyroid gland palpable but not enlarged.	Euthyroid	6·9	—	—	—	—	—	—
12	J. M.	F	25	Clinically diagnosed as mild thyrotoxicosis with moderate enlargement of thyroid gland. Treated with 5 mc. of ^{131}I with doubtful improvement, and no change in the B.M.R. or ^{131}I tracer test 2 months later. Repeat PBI 5·7 µg.%.	? Hyperthyroid	3·0	Plus 22	108 µc.	17·0	54·5	45·0	26·0

TABLE II. SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN 60 PATIENTS—*(continued)*

Case No.	*Patient*	*Sex*	*Age*	*Case Notes*	*Final Clinical Assessment of Thyroid State*	*Serum PBI µg.%*	*B.M.R. %*	*Radioactive Iodine (^{131}I) Tracer Test*				
								Dose	*1 hour Uptake %*	*24 hours*		
										Uptake %	*Conversion Ratio %*	*Excretion %*
13	B. B.	F	26	Slight thyrotoxicosis, with moderate diffuse enlargement of thyroid gland and slight exophthalmos; 18 weeks pregnant; marked clinical improvement on tapazole.	Hyperthyroid	9·1	Plus 24	—	—	—	—	—
14	J. M.	F	33	Thyrotoxicosis with moderate diffuse enlargement of thyroid gland and oedema of eyelids and conjunctivae. Treated with 2·6 mc. ^{131}I 2 months earlier. Thyroid extracts given orally and the pituitary area and the orbits irradiated in view of oedema of the eyes. Has become clinically worse during the last few weeks due to treatment with cortisone for generalized dermatitis.	Hyperthyroid	11·0	Plus 54	92 µc.	50·0	96·0	78·5	12·2
15	Y.	M	—	Plunging thyroid. Diffusely enlarged thyroid gland present after coughing. No evidence of toxicity.	Euthyroid	5·2	—	—	—	—	—	—
16	M. E.	F	6	Pituitary dwarfism. Bone age 4 years. Looks even younger. Plasma cholesterol 189 mg.%.	Euthyroid	5·8	—	—	—	—	—	—
17	E. W.	F	43	Thyrotoxicosis. Toxic nodule in right lobe. Slight exophthalmos. Complete recovery after surgical excision.	Hyperthyroid	9·2	Plus 13	96·5 µc.	35·0	82·5	76·0	?
18	E. C.	M	3	Nephrosis treated with ACTH and cortisone. (Medalie, M. and Bloomberg, B. M., are publishing a complete report on this case.)	Euthyroid	2·8	—	—	—	—	—	—
19	D. L.	F	38	Partial thyroidectomy 7 years ago. Thyroid gland not palpable. Severe iron-deficiency anaemia.	Euthyroid	4·0	Plus 10	—	—	—	—	—
20	L. G.	M	41	Anxiety state. Treated with phenobarbitone with clinical improvement. Thyroid—normal in size.	Euthyroid	5·3	Plus 16	—	—	—	—	—
21	N. L.	M	41	? Thyrotoxicosis. ? Anxiety state. Treated with 5 mc. ^{131}I with improvement.	Hyperthyroid	6·7	Plus 67	99 µc.	60·0	96·0	76·0	7·0

TABLE II: SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN 60 PATIENTS—*(continued)*

Case No.	*Patient*	*Sex*	*Age*	*Case Notes*	*Final Clinical Assessment of Thyroid State*	*Serum PBI* μg.%	*B.M.R.* %	*Radioactive Iodine (^{131}I) Tracer Test*				
								Dose	*1 hour Uptake* %	*24 hours*		
										Uptake %	*Conversion Ratio* %	*Excretion* %
22	A. S.	F	9	Thyrotoxicosis. Moderate diffuse enlargement of thyroid gland with exophthalmos. Plasma cholesterol 146 mg.%. Treatment with tapazole commenced on about 15.11.52. On 26.11.52 PBI 12·3 μg.%. On 3.1.53 PBI 5·4 μg.%.	Hyperthyroid	19·0	Plus 28	—	72·0	—	5·1	
23	E. F.	F	40	Hypertension and nephritis and incipient cardiac failure. Anxiety neurosis. Thyroid normal in size.	Euthyroid	7·7	Plus 24	124 μc.	9·5	40·0	47·0	?
24	T. H.	F	37	Mild diabetes, 1951. Severe diabetes now, plus ? hyperthyroidism. Considerable clinical improvement 3 months after 4 mc. ^{131}I. On insulin treatment.	? Hyperthyroid	8·4	Plus 44	117 μc.	24·0	70·5	73·0	12·2
25	M. L.	F	17	Anxiety neurosis. Mild hypertension. Thyroid gland normal in size.	Euthyroid	6·5	Plus 3	—	—	—	—	—
26	H. H.	F	11	Diffusely enlarged thyroid for 1 year but does not appear toxic and is gaining weight normally for age.	Euthyroid	6·1	Minus 9	—	—	—	—	—
27	S. M.	F	38	Bantu. Thyrotoxicosis. Moderate diffuse enlargement of thyroid gland with exophthalmos and pretibial myxoedema. Plasma cholesterol 100 mg.%. Treated with 5 mc. ^{131}I on 27.11.52. Clinically shows considerable improvement. In February 1953 PBI 4·8 μg.% B.M.R. plus 29%, cholesterol 183 mg.%. (Metz, J. and Meyer, J. have published a complete report on this case—see bibliography.)	Hyperthyroid	11·8	Plus 76	100 μc.	73·0	94·0	71·0	5·5
28	E. S.	F	—	Thyrotoxicosis with moderate diffuse enlargement of thyroid gland.	Hyperthyroid	7·2	—	83 μc.	28·5	84·0	76·5	2·3
29	L. S.	M	9	Chondro-dystrophy. On thyroid extracts for some years with no improvement.	Euthyroid	3·6	—	—	—	20·0	—	74·0
30	A. H.	F	52	History of thyrotoxicosis some years ago. Emotional and excitable. ? thyrotoxicosis. Thyroid gland normal in size.	? Hyperthyroid	7·1	Plus 24	96 μc.	47·5	89·0	85·0	8·2

TABLE II: SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN 60 PATIENTS—*(continued)*

Case No.	*Patient*	*Sex*	*Age*	*Case Notes*	*Final Clinical Assessment of Thyroid State*	*Serum PBI μg.%*	*B.M.R. %*	*Radioactive Iodine (^{131}I) Tracer Test*				
								Dose	*1 hour Uptake %*	*24 hours*		
										Uptake %	*Conversion Ratio %*	*Excretion %*
31	J. K.	F	40	Hyperthyroidism 6 years ago during first pregnancy. Recurred 3 years ago during second pregnancy. Treated with thiouracil during attacks and for last 9 months. Now complains of lassitude. Dry skin and hair. Thyroid gland shows a slight diffuse enlargement.	? Euthyroid	4·5	Minus 20	—	35·8	76·0	68·0	16·7
32	J. C.	M	32	? Mild thyrotoxicosis. Slight diffuse enlargement of thyroid gland with slight exophthalmos. Plasma cholesterol 103 mg.%.	Hyperthyroid	7·8	Plus 22	—	—	—	—	—
33	B. S.	F	38	? Mild hypothyroidism. Treated with thyroid extracts with some clinical improvement.	? Hypothyroid	2·9	Minus 9	—	—	—	—	—
34	S. T.	F	19	Psychoneurosis. Thyrotoxicosis considered in differential diagnosis because of some proptosis. Thyroid gland normal in size.	Euthyroid	6·3	Plus 16	—	—	—	—	—
35	T. J.	F	35	Anxiety state. Does not look toxic, but investigations carried out because of a moderate diffuse enlargement of the thyroid gland.	Euthyroid	3·2	Plus 2	—	—	—	—	—
36	F. C.	F	45	Chronic anxiety state with hyperventilation syndrome.	Euthyroid	4·1	Minus 37	84 μc.	11·2	29·0	38·0	24·2
37	F. O.	F	23	Sheehan's syndrome. (? Postpartum necrosis of pituitary.) Plasma cholesterol 300 mg.%. Urinary follicle-stimulating hormone less than 3 mouse uterine units per 24 hours. Urinary 17-ketosteroids 1·4 mg. per 24 hours.	Hypothyroid	2·2	Minus 32	—	—	—	—	—
38	J. D.	F	38	? Slight diffuse enlargement of thyroid gland, but no obvious evidence of thyrotoxicosis.	? Euthyroid	6·5	—	—	—	—	—	—
39	J. L.	F	45	Hypertension with anaemia. Plasma cholesterol 136 mg.%.	Euthyroid	2·0	Minus 12	94·5 μc.	13·0	42·0	45·0	52·7
40	J. T.	F	33	Tachycardia. Anxiety state.	Euthyroid	2·3	—	—	—	—	—	—
41	R. H.	F	42	? Thyrotoxicosis. Moderate diffuse enlargement of thyroid gland. Tachycardia. In view of low PBI, no therapy given and clinical state remains unchanged with no deterioration.	? Hyperthyroid	0·9	—	—	—	—	—	—

TABLE II: SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN 60 PATIENTS—(*continued*)

Case No.	*Patient*	*Sex*	*Age*	*Case Notes*	*Final Clinical Assessment of Thyroid State*	*Serum PBI μg.%*	*B.M.R. %*	*Radioactive Iodine (^{131}I) Tracer Test*				
								Dose	*1 hour Uptake %*	*24 hours*		
										Uptake %	*Conversion Ratio %*	*Excretion %*
42	A. S.	F	46	Obesity (? Cushing's syndrome) hypertension with congestive cardiac failure. Clinically does not appear myxoedematous. Plasma cholesterol 326 mg.%.	? Euthyroid	1·9	—	—	—	—	—	—
43	R. M.	F	32	Non-toxic adenoma of thyroid.	Euthyroid	5·5	—	94 μc.	9·2	39·0	40·0	57·1
44	C. N.	F	38	? Thyrotoxicosis.	? Hyperthyroid	5·7	Plus 29	—	—	—	—	—
45	H. L.	F	56	Angina pectoris with coronary atherosclerosis. Postmenopausal. Thyrotoxicosis suspected because of tachycardia. Thyroid gland normal in size.	? Hyperthyroid	5·7	Plus 20	—	—	—	—	—
46	I.	F	49	Slight thyrotoxicosis with moderate diffuse enlargement of thyroid gland and exophthalmos. Treated with tapazole with considerable clinical improvement.	Hyperthyroid	8·2	—	—	—	—	—	—
47	L. M.	F	—	Thyrotoxicosis.	Hyperthyroid	8·8	—	—	—	—	—	—
48	M. B.	F	48	Patient's appearance suggested mild hypothyroidism; not confirmed by investigations. Plasma cholesterol 170 mg.%.	? Euthyroid	7·9	Plus 2	—	—	—	—	—
49	N. N.	F	32	Thyrotoxicosis with moderate diffuse enlargement of thyroid gland. 5-6 months pregnant. Thiouracil treatment resulted in considerable clinical improvement.	Hyperthyroid	12·0	Plus 55	—	—	—	—	—
50	E. R.	M	28	Thyrotoxicosis in 1951 (B.M.R. + 24%). Treated with 9·2 mc. ^{131}I in 2 doses. Difference of opinion as to present clinical assessment of thyroid state. Thyroid gland appears to be normal in size.	? Euthyroid	4·4	Minus 20	79·5 μc.	17·0	62·5	54·0	40·4
51	R. F.	M	21	Thyrotoxicosis with slight diffuse enlargement of thyroid gland and slight exophthalmos. Plasma cholesterol 96 mg.%.	Hyperthyroid	8·4	Plus 43	—	—	—	—	—
52	B. W.	F	18	? Anxiety state. Thyroid gland appears to be normal in size. Thyrotoxicosis considered but B.M.R. (done elsewhere) was normal and PBI normal. No treatment given and clinical state has remained unchanged with no deterioration.	? Euthyroid	6·5	—	—	—	—	—	—

TABLE II: SERUM PROTEIN-BOUND IODINE ESTIMATIONS IN 60 PATIENTS—(*continued*)

Case No.	*Patient*	*Sex*	*Age*	*Case Notes*	*Final Clinical Assessment of Thyroid State*	*Serum PBI μg.%*	*B.M.R. %*	*Radioactive Iodine (^{131}I) Tracer Test*				
								Dose	*1 hour Uptake %*	*24 hours*		
										Uptake %	*Conversion Ratio %*	*Excretion %*
53	L. B.	F	15	Thyrotoxicosis with moderate diffuse enlargement of thyroid gland and slight exophthalmos.	Hyperthyroid	10·2	Plus 27	85 μc.	37·0	81·0	83·0	8·0
54	M. P.	F	17	Moderate diffuse enlargement of thyroid gland. Patient lives in N.E. Transvaal. ? Endemic goitre. No evidence of thyrotoxicosis.	Euthyroid	5·9	Minus 3	—	—	—	—	—
55	M. D.	F	12	Hypothyroidism. Mentally retarded. X-ray shows osteochondral hypothyroidism. Osseous age 8 years. Typically myxoedematous appearance. Plasma cholesterol 315 mg.%.	Hypothyroid	1·1	—	—	—	—	—	—
56	V. D.	M	57	Myxoedema with acute coronary thrombosis. Thyroid gland appears to be normal in size. Plasma cholesterol 220 mg.%.	Hypothyroid	1·8	Minus 20	—	—	—	—	—
57	J. W.	F	31	Thyrotoxicosis treated with 4 mc. ^{131}I on 13.11.52. Clinically improved, but still appeared to be mildly toxic when PBI test was done on 27.1.53, and thyroid gland showed a slight diffuse enlargement.	Hyperthyroid	7·0	Plus 15	88 μc.	14·0	37·0	46·0	42·8
58	M. K.	F	45	21.8.52. Sub-total thyroidectomy for toxic nodular goitre. Marked clinical improvement with no evidence of toxicity when PBI and B.M.R. studies carried out.	Euthyroid	2·5	Minus 14	—	—	—	—	—
59	C. B.	F	—	Cardiac damage. On propylthiouracil until 2 months ago. Thyroid gland shows a moderate diffuse enlargement but there is no evidence of thyrotoxicosis.	Euthyroid	5·6	Plus 16	—	—	—	—	—
60	J. R.	F	37	Thyrotoxicosis with a considerable diffuse enlargement of thyroid gland and exophthalmos.	Hyperthyroid	21·5	Plus 71	—	—	—	—	—

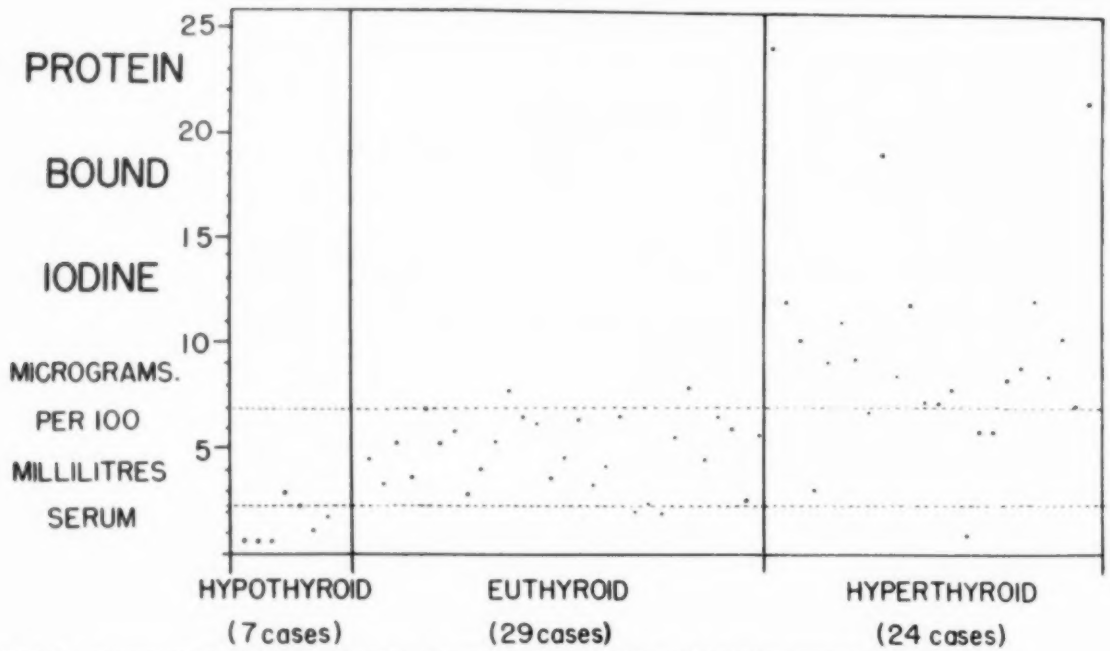

FIG. 2. Correlation of Serum Protein Bound Iodine with Thyroid state assessed clinically.

Dotted lines indicate extremes of P.B.I. range found in 10 normal subjects. (TABLE I)

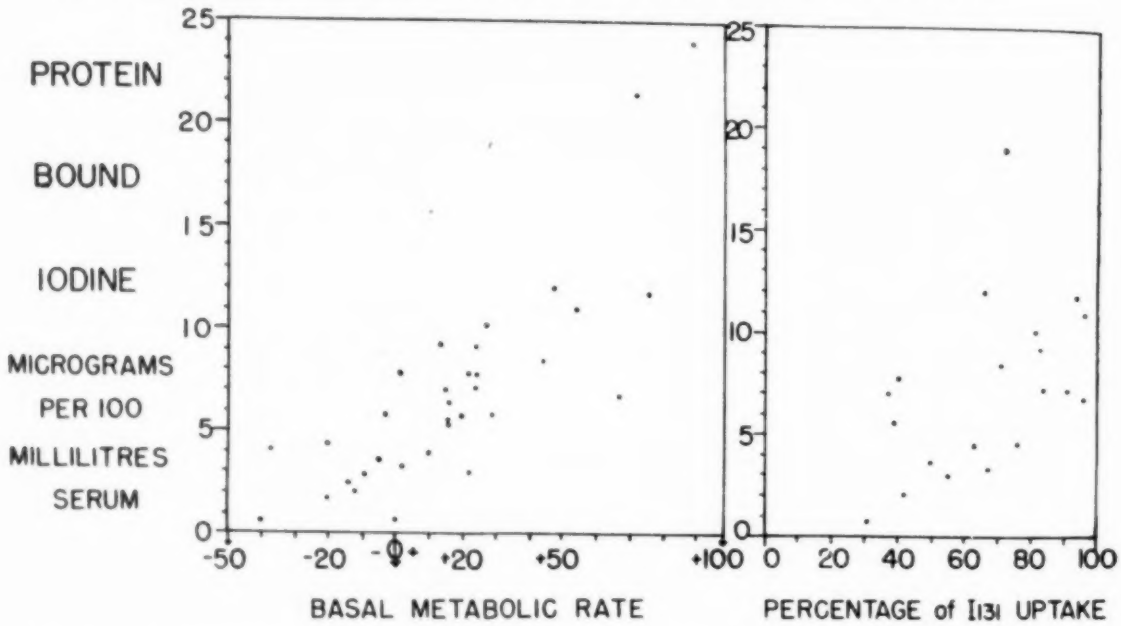

FIG. 3. Correlation of Serum P.B.I. with Basal Metabolic Rate and Radio-Iodine Uptake.

TABLE III: SUMMARY OF SERUM PROTEIN-BOUND IODINE RESULTS IN 46 PATIENTS

Final Clinical Assessment of Thyroid State	*Number of Cases*	*Serum PBI μg. %*	
		Range	*Mean*
Hypothyroid	5	0·6–2·2	1·3
Euthyroid	23	2·0–7·7	4·7
Hyperthyroid	18	6·7–24·0	11·3

results indicate a reasonable correlation of serum PBI levels with the final clinical diagnosis. Figure 3 also shows a reasonable relationship of serum PBI levels with the B.M.R. determinations, considering that these estimations are fundamentally quite different. In Table III a summary is presented of the findings in 46 of the patients shown in Table II. The results in 14 patients where the clinical diagnosis was in doubt, and in whom the final clinical assessment of the thyroid state is queried in Table II, have been omitted.

(To be concluded)

PLACENTA SUCCENTURIATA AS A CAUSE OF ANTE-PARTUM HAEMORRHAGE

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As ante-partum haemorrhage due to placenta succenturiata appears to be uncommonly recorded in the literature, a case is described which presented as an ante-partum haemorrhage of some severity.

CASE REPORT

A Bantu woman, about 18 years of age, was admitted to the Maternity Unit, Harari African Hospital, Salisbury, at 11 a.m. on 16 November, 1950. She gave a history of intermittent vaginal bleeding since 11 November with vague abdominal pains; she had missed nine menstrual periods but did not know the date of her last menstrual period. She was a primipara, had felt well up to the commencement of bleeding, and had not had any medical attention until admission. The woman stated that she had not felt any foetal movements since the day after the haemorrhage had begun but she was vague as to how much blood she had lost.

Examination on admission showed a young Bantu woman, extremely shocked, with very pale mucous membranes, pulse of poor volume, rate 140 per minute, blood pressure 70/30, temperature 97.6° F with rapid respirations. Abdominal palpation revealed a 40 weeks' pregnancy, breech right sacro-anterior, with no foetal heart heard. There was nothing abnormal in the chest except a generalized systolic murmur heard over the cardiac area. From the history and examination a diagnosis of placenta praevia was made and treatment for shock and haemorrhage was instituted.

Blood-grouping showed her blood to be Group A and a haemoglobin estimation made at this time by a Sahli haemoglobinometer was 25%. She was given morphine sulphate gr. ¼ (15 mg.) and dextran solution was given intravenously until compatible blood could be obtained. One pint of dextran, 2 pints of Group O and 1 pint of Group A compatible blood were given, the first rapidly and the others more slowly. During this time the patient drank freely and slept, and her condition steadily improved; there was no further haemorrhage vaginally, the pulse rate fell to 90 per minute and the blood pressure rose to 100/60 by 8 a.m. on 17 November.

On that day at 8.15 a.m. a vaginal examination was made and a 2-finger os was felt with placental tissue covering the whole of the os. On finding this no further examination was attempted. As no foetal heart could be heard it was decided to give a further pint of compatible blood and leave the patient until her improved condition had stabilized (she had been lying in a grass hut for 5 days previous to her admission). During the giving of this further pint of Group A blood, a brisk vaginal haemorrhage set in and a Caesarean section was decided on.

A classical Caesarean section was carried out at 10.20 a.m. under general anaethesia. When the uterus was opened the incision on the anterior wall revealed the placenta lying underneath. This was pushed aside and a macerated male foetus, weight 7 lb. 3 oz., was delivered. The placenta was next removed but the membranes could not be easily delivered. It was then seen that a small lobe of placental tissue was on the lower segment entirely covering the internal os. This was removed by blunt dissection as it was rather adherent at one point, which resulted in considerable haemorrhage. The uterus was then sutured in layers and the abdomen closed. During the operation a further pint of dextran and a pint of Group A blood was given. At the end of the operation the patient's condition was reasonable.

The patient was given soluble penicillin in 100,000 unit doses 6 hourly for 2 days, followed by 1 c.c. procaine penicillin (Glaxo) twice daily for 3 days. Her post-operative convalescence was uneventful apart from a slight bronchitis from 20 to 22 November. She was discharged from hospital on 4 December, general condition good, haemoglobin (Sahli) 90%, abdominal wound well healed. Seen post-natally on 17 January 1951 she said she felt well and nothing abnormal could be found.

COMMENT

The history and clinical examination of this patient in every way pointed to a diagnosis of placenta praevia. The unusual severity of the haemorrhage suggested a placenta praevia of Type III or IV and the recurrence of the bleeding after the transfusion of the blood prompted the carrying out of a Caesarean section, when the placenta was revealed as being on the anterior uterine wall. Because of

the history and the fact that placental tissue was felt vaginally it was thought that there must be some portion of the placenta in the lower uterine segment. The discovery of a succenturiate lobe in this position revealed the cause of the severe ante-partum haemorrhage despite the presence of the main placental mass on the anterior wall of the uterus. Kerr and Moir (1949) point out that in placenta succenturiata, post-partum haemorrhage may occur if the succenturiate lobe is left behind but they do not mention this condition as a cause of ante-partum haemorrhage. Siegler and Sacks (1941) describe a case of placenta praevia with placenta succenturiata which presented as an ante-partum haemorrhage, where the diagnosis was made by Ceasarean section. They reported that they had not heard of a similar case but Torpin and Hart (1941) published a series of cases of placenta bilobata in which several presented as ante-partum haemorrhage.

The shocked state of the patient on admission was probably due to a journey of some 50 miles which she had made in an old lorry from a Native Reserve. On the way to hospital she may have lost a considerable amount of blood but her relatives were very vague about the whole affair and no satisfactory explanation could be obtained as to why they were so long in bringing the patient to hospital.

Thanks are due to Dr. D. M. Blair, *O.B.E.*, M.D., Acting Secretary for Health, Southern Rhodesia for permission to publish this case.

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TORSION OF A PYOSALPINX

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Since the advent of chemotherapy pyosalpinx has become a disappearing disease and so the occurrence of its complications less frequent. A case in which torsion occurred is here recorded. Tubal infection is almost invariably bilateral and an acute state, unless adequate treatment is instituted, lapses into a chronic condition. In this case symptoms and signs of an emergency arising from a chronic condition were present. Torsion of a pyosalpinx is rare because in nearly all cases the tubes become adherent and fixed to surrounding structures, particularly omentum and intestine: in fact, on this account removal of a pyosalpinx may be fraught with the greatest difficulty and tax the skill and dexterity of the most experienced operator.

A. D., aged 21 years, an African negress, was admitted to Edenvale Hospital on 13 April 1953.

Complaint. Lower abdominal pain for the past 2 days.

History. The pain was of sudden onset, at first cramplike, later becoming constant and more severe; did not radiate and much worse on the left side; there was some nausea at the onset. For several months past there had been a thick vaginal discharge and burning on micturition but no frequency; there had been no recent exacerbation of these latter symptoms. The bowels had acted twice spontaneously since the onset of the pain.

Menstrual History. Menarche at 13 years of age; katamenia regular, 5/28; no dysmenorrhoea. The last menstrual period had commenced on 8 February 1953, i.e. 9 weeks previously. Nine days before, slight vaginal bleeding started and lasted for 2 days.

Obstetric History. In March 1952 she had been delivered by Caesarean section of a full-time living child: no details were available as to what was the indication for abdominal delivery, but it was probably for cephalopelvic disproportion.

CLINICAL FINDINGS

A well-nourished adult negress who looked acutely ill. No pallor of the oral mucosa or conjunctivae. Temperature 99.8° F. Blood pressure 130/75 mm. Hg. Pulse—full volume, rate 120 per minute. There were no abnormal findings in the central nervous, cardio-vascular or respiratory systems.

Abdomen. No distension or visible peristalsis: a firmly healed transverse incision at the level of the anterior superior iliac spines: the upper abdomen moved freely with respiration. On palpation there was slight tenderness in the right iliac fossa and marked tenderness in the left iliac fossa. There were no palpable tumours and no external herniae.

Vaginal Examination. Per speculum was seen a nulliparous cervix with a small congenital erosion of the external os. There was some thick yellow discharge in the vagina but no reddening of the external urinary meatus or Sanger's macules.

On bimanual examination was felt:

(1) a small anteverted uterus, attempts to move which caused agonizing pain;

(2) in the pouch of Douglas, a sharply defined mass, the size of a golf ball and the consistency of india rubber, which was not tender;

(3) on the left side, above and behind the uterus, an exquisitely tender mass, the size of a tennis ball and seemingly fixed to the posterior wall of the pelvis.

The urine showed no abnormal constituents.

DIAGNOSIS

In the differential diagnosis the following conditions were considered:

(1) A lesion of the alimentary tract, e.g. perforation, occlusion or thrombosis: these were excluded because of the absence of vomiting, the pain had remained localized, the bowels had acted since the onset of symptoms and there was no rigidity or abdominal distension.

(2) A urinary tract lesion: this was excluded because the pain had remained localized and no abnormal constituents were found in the urine.

(3) A tumour of each ovary with torsion of that on the left side: this would not account for the amenorrhoea,

vaginal discharge or dysuria, but was otherwise in accord with the history and findings.

(4) Some complication of pregnancy: the amenorrhoea suggested a threatened abortion or ectopic pregnancy. The former was excluded because of the sudden onset, severe pain, tenderness and absence of violet colour of the vagina and cervix uteri. Ectopic pregnancy was considered more likely because of the nature of the pain and the tender mass on the left side: against this assumption was the fever, vaginal discharge and dysuria.

(5) An infective condition of the uterine adnexa: this was considered likely because of the dysuria and vaginal discharge, the masses in the pelvis, the non-pregnant state of the uterus and the strictly localized pain and tenderness. Furthermore the patient had been admitted by the casualty officer 6 hours previously and the following treatment was then prescribed: penicillin 500,000 units 6-hourly, sulphatriad 2 4-hourly and pethidine 100 mgm. statim. More important, a ½-hourly record of the pulse rate had been ordered, and this showed that the rate had increased steadily from 100 per minute to 120.

TREATMENT

Laparotomy was decided on.

General anaesthesia was induced with pentothal and maintained with nitrous oxide, ether and oxygen. The bladder was emptied by catheter and a bimanual examination confirmed the previous findings. The patient was arranged in the dorsal position.

The abdomen was entered through a lower midline incision. A small quantity of sero-sanguineous fluid was found in the peritoneal cavity. There was no matting of the intestines or omentum. The uterus was of normal size and lying behind it in the pouch of Douglas was the right Fallopian tube. The isthmus was of normal thickness and length but the ampulla was the size of a golf ball, which gave the tube a retort shape. The site of the abdominal ostium was marked by a dimple on its lower lateral aspect; the surface was smooth and non-adherent to neighbouring structures. On the left side a similar state of affairs was found except that the ampullary end of the tube was the size of a tennis ball and it was not possible to identify the abdominal ostium. At the junction of the isthmial and ampullary portions, the proximal end of the tube had rotated through three-quarters of a circle in an antero-posterior direction. The ampullary end was plum-coloured and congested and had subserous petechial haemorrhages and flakes of lymph on the surface. On each side the ovary appeared normal, was separate from the tube and was in normal relation to the broad ligament; a shrunken corpus luteum was seen on the right ovary.

On each side the meso-salpinx was divided between clamps and ligatured and the tubes resected at their cornual junctions. The abdomen was closed without drainage in three layers.

After-treatment. Penicillin 200,000 units 4-hourly for 5 days, streptomycin 0.5 gram twice a day for 5 days, and omnopon 1/6 gr. 6-hourly for 48 hours. Rising and ambulation commenced 12 hours after operation. The patient made an uninterrupted recovery and was discharged home on the 12th day after admission.

PATHOLOGY

On each side the isthmial portion showed a slightly thickened wall. Each ampullary portion was spherical and showed a uniformly thickened wall ⅜ inch thick: the enlargement was considerably greater on the left side. Each ampullary portion contained a smooth-walled cavity without any sign of plication, which was filled with a thin yellow non-odorous pus strongly suggestive of gonococcal infection. On each side a constriction separated the lumen of the isthmus from the cavity of the ampullary portion. Bacteriological examination revealed the pus to be sterile. There was no macroscopic evidence of tuberculous infection of the tubes or ovaries. The specimens were sent for microscopic section, but unfortunately were lost in transit.

DISCUSSION

This case presented various interesting features.

The infection must have been contracted since the Caesarean section one year previously.

From the history and appearance of the tubes and pus, the condition was almost certainly a sequel of gonococcal infection.

The infection must have spread upwards and had been arrested at the abdominal ostium. Presumably a small amount of oedema in the acute stage had sealed the ostia on each side: at a later stage the tubal lumen at the junction of isthmus and ampulla had been occluded.

The ovaries did not appear to have been infected and there was a complete absence of pelvic adhesions.

There was no evidence of a recent pregnancy nor any explanation for the history of amenorrhoea.

The only symptoms of previous pelvic infection were vaginal discharge and dysuria.

With the torsion there arose acute symptoms. The strictly localized pain, tender mass on the left side and increasing pulse rate were considered to call for laparotomy.

After the pelvis was explored the line of action was obvious. Both ovaries appeared healthy and were conserved. Both tubes were diseased and obviously functionless and were therefore removed. Many authorities consider it wise to remove the uterus when doing a salpingectomy for infected tubes: this must add to the magnitude of the operation, and unless there is some lesion of the uterus calling for its removal it is doubtful whether it is ever justified, especially in a young woman. This patient is a member of a race that attaches great importance to menstruation and fertility: unfortunately her fertility had been destroyed but from the history there did not seem to have been any interference with the menstrual rhythm and so hysterectomy was not even considered. Among these people, anything in the way of treatment which impairs fertility or suppresses menstruation will merely bring Western medicine into disrepute and should be avoided, even if it means leaving the woman with symptoms.

SUMMARY

The history and clinical findings in a case of torsion of a pyosalpinx are recorded. The findings at operation are described. The differential diagnosis is discussed. The details of operation, after-treatment and convalescence are recorded.

I am indebted to Dr. L. Feitelberg, Medical Superintendent of Edenvale Hospital, for his kind permission to publish this case.

AN UNUSUAL CASE OF ACCIDENTAL UTERINE HAEMORRHAGE

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Premature separation of the normally situated placenta with a Couvelaire uterus ('uteroplacental apoplexy') is a serious but fortunately a rare condition. Most frequently found in accidental haemorrhage of the concealed type, it is often associated with pre-eclamptic toxaemia or essential hypertension. The latter conditions need not be present however, and a cause for separation of the placenta is then seldom found. Bleeding is then incidental. Rarely trauma such as external cephalic version, a fall or blow on the abdomen, strain, coitus, purging, enemata, severe coughing, and even turning in bed, is blamed.

The following typical symptoms and signs are well known:

(1) *Bleeding*, external, internal or most frequently combined.

(2) *Abdominal pain*, more often than not followed by the onset of labour pains, which usually are weak.

(3) *A tender uterus* with varying degrees of hardness, associated with the retention of blood within its cavity, and extravasation of blood into the broad ligaments (Davis and McGee [1]). Absence of pain is not unknown, especially in minor degrees of placental separation. The uterus is exceptionally flabby and dilated (De Lee [2]).

(4) *Pre-eclamptic toxaemia or essential hypertension* is present in the vast majority of cases. Neither of these conditions was present in 5.9% of Harrar's [3] cases, and in 33.5% of Holmes [4] cases. Polak [5] blamed trauma in 18.7%, and Davis and McGee [1] in 1.8% of 164 cases. Goethals,[6] who operated on 12 patients with accidental haemorrhage, found a Couvelaire uterus in 2 without toxaemia.

(5) *Shock* is very frequent, out of all proportion to the amount of blood lost. A normal pulse however, does not exclude premature separation of the placenta (Portes [7]).

(6) *Disappearance of the foetal heart-sounds* is common, and consequently foetal mortality is as high as 60–90% (Irving [8]).

The following case is reported because of the unexpected findings at operation.

CASE

Mrs. V. M., aged 27 years, 36 weeks pregnant, was admitted to hospital with vaginal bleeding which had started 5 hours previously.

Obstetric history. No previous pregnancy.

Medical and surgical history. Nothing of note.

Menstrual history. Regular with normal loss, 3–4/28 type. No dysmenorrhoea. Last normal period, 22 February 1952. Expected date of confinement, 30 November.

Antenatal history (obtained from patient's doctor's notes). Regular monthly attendance from the 3rd month of pregnancy. On 3 August, blood pressure 90/80; 100/80 on 7 September; and 120/80 on 6 October. Weight remained normal until the last month when a 9 pounds gain was recorded. Urine normal. Abnormal bruising or bleeding had never occurred.

Present history. Patient was awakened at 3 a.m. on 21 October by mild lower abdominal cramp-like pains. As she had often done before, she gave herself an enema, because 'of a feeling of being constipated'. Immediately afterwards fairly profuse bright-red vaginal bleeding took place, and the pains in her abdomen persisted at irregular intervals. Her own doctor was consulted, and he reported severe bright-red bleeding with large clots, blood pressure of 110/60, and a pulse rate of 80 per minute. The foetal heart-sounds were present, and the foetal head was 'in the pelvis'. Admitted to hospital 6 a.m.

Condition on Examination

Blood pressure 110/80. Pulse 80 per minute. Temperature 98. Slight pallor. General condition surprisingly good, considering the reported blood loss. No oedema.

Abdominal examination. Fundus uteri 3 fingers down from the xiphisternum. Irregular poor contractions, with no tenderness or rigidity. Foetus easily felt, lying in the L.O.A. position. Head fixed. Foetal heart-sounds, 160 and regular.

Rectal examination. Foetal head in the pelvis. Cervix unprepared, the external os admitting tip of finger. Fair amount of dark-red loss vaginally. Membranes intact.

Potassium bromide and chloral hydrate was administered with good effect.

4 p.m.: Poor contractions. Continuous not inconsiderable dark-red loss. No further progress in dilatation of cervix. Foetal heart-sounds 120–140, and regular. Maternal pulse 80, blood pressure 110/60.

Blood investigation. Group A, Rhesus positive (Rho). Haemoglobin 11.8 mgs. Kahn, Rappaport and Wassermann tests negative.

Treatment

Because of the persistent blood loss and absence of progress, operation was decided upon. Anaesthesia (Dr. J. Smith) at 5 p.m.: pentothal, flaxedil, gas and oxygen.

A large quantity of free fluid, heavily blood-stained, was seen on opening the abdomen. The uterus showed considerable extravasations of blood under the serosa on both anterior and posterior surfaces. Both broad ligaments and the peritoneum over the bladder and anterior abdominal wall were similarly involved. The lower uterine segment was poorly developed. A transverse incision into the lower segment revealed a large amount of old blood, and the liquor amnii was markedly blood-stained. The foetus cried immediately on extraction, and the placenta lying almost free in the fundus was spontaneously expelled immediately afterwards, with a large retroplacental clot. The uterus contracted well, and there was less than normal postpartum loss.

Blood transfusion (1 pint) followed by intravenous dextrose in water (1 pint) were administered. The patient's condition remained excellent throughout.

Infant. Premature. Weight 5 lbs. Cried well after delivery. Progress uneventful.

Placenta. Smaller than normal. No evidence of infarction. A large retroplacental clot present, measuring exactly 1 pint.

Puerperium. Uneventful. Blood pressure varied between 100/60 and 120/70 until discharge from hospital. Six weeks later there was no alteration in the blood pressure. The urine was clear.

SUMMARY

An unusual case of a Couvelaire uterus unassociated with pre-eclamptic toxaemia or essential hypertension, in the almost entire absence of physical signs, with the delivery of a living child, by Caesarean section.

DISCUSSION

Etiology. Vaginal bleeding immediately following the administration of an enema suggested trauma as the exciting cause in the case described. This, however, may only have been an incidental factor, as labour appears to have already started before the enema was given. No other factor could be blamed for the bleeding. Was the 9-pound gain of weight a premonitory sign of impending toxaemia in the last month of the pregnancy in the absence of hypertension?

Dieckmann [9] suggests that there are a group of patients who have abnormalities in bleeding- and clotting-times of their blood. When they become pregnant a bleeding tendency with abruptio placentae, eclampsia and severe haemorrhage may develop.

Weiner *et al.*[10] report a defective clotting mechanism in certain patients, in whom a dead erythroblastotic foetus was retained in utero, due to there being little or no fibrinogen. They suggest, however, that coagulation defects follow rather than precede, and are restricted to placental separation of severe type, associated with severe blood loss. Blood defects are not manifest in mild cases. They therefore advocate blood transfusion with at least 1500 c.c of fresh blood in severe cases of premature separation of the normally-situated placenta to adequately replace deficient fibrinogen.

Diagnosis

In the absence of hypertension the following were considered:

(1) *Accidental haemorrhage (premature separation of the normally-situated placenta).* The nature of the bleeding, the premature labour and its almost immediate onset, and the presence of a fixed foetal head, suggested accidental haemorrhage of a mixed mild type. Complete absence of abdominal signs and the unexpected findings at operation were difficult to explain.

(2) *Placenta praevia.* The deciding factor which warranted exclusion of this condition was the fixed foetal head. Browne [11] however describes a case of low implantation of a thin membranous placenta over the internal os in a primigravida, with a fixed foetal head. The nature of the haemorrhage, too, was unlike that of placenta praevia.

(3) *Rupture of the marginal sinus.* In recent years a large volume of the American literature has been devoted to this subject. Fish and co-authors [12] indeed maintain that rupture of the marginal sinus was responsible for a third of all cases of vaginal bleeding in the last trimester! The placenta reveals a rent in the marginal surface, with an old or recent blood clot adherent to the marginal sinus, occasionally over a narrow surface of the maternal part of the placenta. Briefly, the features shown are painless vaginal bleeding occurring near term or in labour, with a greater tendency to bring on premature labour than placenta praevia and a low foetal mortality (4.5%).

(4) *Bleeding from a circumvallate placenta.* Blood loss in this condition arises from a small separation of the placenta, or more probably from a rupture of the marginal sinus. Hobbs and Price [13] discovered bleeding from this cause before delivery in 1 in 138 cases. Hunt, Mussey, and Faber [14] found 1 in 188 deliveries, and Paalman and Vander Veer [15] 14 cases in 3 years (1 in 208 deliveries). Williams [16] described hydrorrhoea gravidarum as a common finding prior to confinement in patients with circumvallate placentae. Paalman [15] in 26.8% of his cases found a watery vaginal discharge, with or without blood loss, with premature labour as high as 37%, and a foetal loss of 29.2%. Death of the foetus was due to a placental deficiency or restriction of growth of the placental plate with inability to meet the demands of the growing foetus. Hunt [17] found a recurrence of circumvallate placenta in subsequent pregnancies in 20% of his cases, with repeated premature labour in some cases.

(5) *Bleeding from the cervix*, due to a polypus, erosion, carcinoma and the like.

SUMMARY AND CONCLUSIONS

A case of accidental haemorrhage (premature separation of a normally situated placenta) is described. Shock and abdominal signs were absent, and yet at Caesarean section a Couvelaire uterus of a severe degree was found, with a large retroplacental clot, and massive haemorrhage into the broad ligaments and abdominal peritoneum. A living child was delivered.

A cause for the premature separation was not found. The possibility of trauma as an exciting cause was considered, in view of the administration of an enema immediately beforehand.

My thanks are due to Dr. B. Seftel for his detailed antenatal notes on the above case, and his assistance.

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THE BENEVOLENT FUND

The following contributions to the Benevolent Fund during July 1953 are gratefully acknowledged:

Votive Cards in Memory of:

Dr. Louis Zinober *by* Dr. Morris J. Cohen.
Mrs. Robert Wolff *by* Dr. and Mrs. W. F. Dickie-Clark and Mr. R. B. Watson.
Dr. E. W. D. Swift *by* Dr. J. P. Immelman.
Mr. A. A. Jurgens *by* Dr. and Mrs. J. Hotz, and Dr. M. Meyers.
Marigold Marais *by* Dr. and Mrs. F. O. Fehrsen.
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Mr. W. Dickie-Clark *by* Dr. Robert Wolff.
Dr. Karl Bremer *by* Dr. and Mrs. A. H. Tonkin, Dr. and Mrs. A. W. S. Sichel, Dr. Vernon Brink, Dr. and Mrs. J. S. du Toit, Dr. P. W. J. Keet, Dr. and Mrs. R. Schaffer, Dr. G. T. van der Vyver, Dr. L. I. Braun, Dr. E. G. van Hoogstraten, Dr. and Mrs. P. F. H. Wagner, Dr. James Black, Dr. Peter Allan, Dr. H. O. Hofmeyr, Dr. W. H. Opie, Dr. J. C. Gie, Dr. A. T. F. Maske, Mr. and Mrs. H. Boehmke, Mr. T. Lindsay Sandes.
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Mr. C. O. Boyle *by* Dr. A. W. Spratt.
Lt.-Com. Quentin Bullard *by* Dr. H. O. Hofmeyr

Total Amount Received from Votive Cards	£36	9	0

Services Rendered to:

Baby Spangenberg *by* Dr. P. J. Rademan.
The late Mrs. Peter Allan *by* Drs. Vernon Brink, H. G. O. Owen-Smith and J. M. Cook
Dr. P. J. Eksteen *by* Mr. F. P. Fouche and Dr. F. J. Durham.

Total Amount Received from Services Rendered	£22	1	0

Donations:

By Members of the Cape Western Branch (Collection Box)	£3	6	10
Dr. R. H. Johnson		10	6
	£62	7	4

IN MEMORIAM

DR. KARL BREMER

Born in April 1885, at Hopefield, C.P., of parents who had come from Germany a few years previously, he spent his early childhood in this town. When he was only 8 years old his father, also a medical practitioner, died at the age of 45 years. Three years later his mother moved, with the family of 7 children, of whom he was the youngest, to Wellington, where she gave music and German lessons. Here he received his schooling. Later he went to the Huguenot College in the same town for the first courses of his B.A. degree.

At the beginning of 1903 he proceeded to Victoria College, Stellenbosch, for the last year before graduating B.A. with honours in Botany, for which he was awarded a Queen Victoria bursary. This enabled him to go overseas for his medical studies—something which before then had seemed impossible. As there were 9 months before the course started in England he went to America with Dr. Bertha Stoneman and began his studies in anatomy and physiology at Cornell University, Ithaca.

In 1904 he studied medicine at St. Bartholomew's Hospital, London, completing his course in 1909. Besides medical and surgical housemanships at St. Bartholomew's he held an appointment at the Great Ormond Street Hospital for Children. Although wishing to stay for further training he was compelled to return to South Africa as his mother had been taken seriously ill at Cradock, C.P. There he started in general practice and continued it until 1915 when answering an appeal by General Botha for doctors in East Africa he joined the S.A.M.C. and served as a Captain for the rest of the war.

Following this he was appointed as the first Medical Inspector of Schools in the Cape Province (Dr. C. L. Leipoldt at that time held a similar appointment in the Transvaal). With only one nurse to help him he visited the schools in the Cape and did much pioneer work in this field. Through his efforts at this time a Child Welfare Association was started at Graaff-Reinet. While still holding this appointment he did much good work in Cape Town during the 'flu epidemic of 1918, assisting in treating patients until he became severely ill himself. Upon his recovery he went into general practice in Graaff-Reinet in February 1919 in partnership with Dr. J. van Schalkwyk.

On 2 occasions, during the following 12 years (1922 and 1930) he went overseas for further study, especially in ear, nose and throat work, and it was in this field that he practised as a specialist in Cape Town from 1931 until 1947, when he retired from active practice on account of his health.

Although not particularly interested in politics in earlier years, he became chairman of the local branch of the Nationalist Party in Graaff-Reinet and was elected as member of the Provincial Council for that constituency in 1920. In 1924 he was elected to Parliament as member for Graaff-Reinet. Finding that this interfered with his practice he resigned his seat in the following year, but was again asked to contest the seat in 1929, when he was once more elected, to remain the member for this constituency until 1943, when he contested and won the Stellenbosch seat.

Towards the end of 1947 he had an attack of cerebral spasm, causing a temporary aphasia, and as a result he reluctantly decided to bid farewell to politics. He found it difficult to adapt himself to an inactive life and his unhappiness was aggravated by the knowledge that as his party had been returned to power he would almost certainly have been given a seat in the Cabinet.

Shortly after this he was made Chairman of the Diamond Board, but resigned the post to become a Senator in December 1948.

When Dr. A. J. Stals died in 1951 Dr. Malan asked him to join his Cabinet as Minister of Health and Social Welfare. This offer he gladly accepted and started his work with characteristic enthusiasm. Shortly after becoming Minister he won the Ceres seat with an increased majority and in the recent general election contested and won the Vasco seat for the Nationalist Party.

Dr. Bremer had been an active member of the Medical Association of South Africa. While in practice in Graaff-Reinet he became President of the Cape Eastern Branch, and in 1937 he was President of the Cape Western Branch. He was a member of the Federal Council of the Association from 1932 to 1939 and on more than one occasion he presided over Federal Council meetings.

From 1935 he was a member of the South African Medical Council on appointment by the Union Government, and in 1943 he was elected President of the Medical Council, a position which he held for 7 years with great distinction until he resigned on becoming Minister of Health in 1951.

From 1928 to 1945 Dr. Bremer was Chairman of the Council of Public Health, appointed under the Public Health Act 1919. He served on many committees and commissions dealing with health matters, notably one on medical education.

He will be remembered by many doctors as a lecturer at the University of Cape Town on Methods of General Practice, a post he held for more than 10 years. Among the honours he received were 2 honorary degrees, an M.D., from the University of Pretoria, and a LL.D. from the University of the Witwatersrand.

For a number of years he was a member of the Board

of Curators of the National Art Gallery, Cape Town, and in many ways helped South African and overseas artists. In 1937 he was elected to the Board of Directors of the S.A. Mutual Life Assurance Society and at the time he became a Minister he was Vice-Chairman of that Board.

Although he never excelled at sport himself, he loved outdoor life and often took part in some sport. He was keenly interested in the accomplishments of Springbok sides or of individual Springboks.

Dr. Bremer married Miss Alice MacKenzie in October 1911 by whom he is survived. His 2 sons (Prof. J. K. Bremer and Dr. P. M. Bremer) are both medical men, and of his 2 daughters (Mrs. van der Merwe of Vryheid, Natal, and Mrs. Gericke of George) the former is the wife of a medical man.

MEDICO-LEGAL

ERRATUM

In the summary of the Bill to amend the Medical, Dental and Pharmacy Act, page 748, of the *S.A. Medical Journal*, 29 August 1953, it was stated: 'This section as amended no longer applies to medical practitioners but only to dentists and druggists.' The words 'dentists and druggists' should have read 'chemists and druggists'.

IN PASSING

UNION OF SOUTH AFRICA : DEPARTMENT OF HEALTH

BULLETIN NO. 33 OF 1953, FOR THE 7 DAYS ENDED THURSDAY, 13 AUGUST 1953

PLAGUE

Nil.

SMALLPOX

Nil.

TYPHUS FEVER

Nil.

EPIDEMIC DISEASES IN OTHER COUNTRIES

At date of latest available information there existed:

Plague: Nil.

Cholera in Calcutta (India); Moulmein (Burma).

Smallpox in Bombay, Calcutta, Madras, Nagapatinam, Tiruchirappalli (India); Haiphong, Saigon-Cholon (Vietnam); Phnom-Penh (Cambodia).

Typhus Fever: Nil.

REVIEWS OF BOOKS

MEDICAL JURISPRUDENCE

Medical Jurisprudence. By Dr. I. Gordon, Dr. R. Turner, and Dr. T. W. Price. (Pp. 944 + lv, with 143 figures. Third Edition. 75s.) Edinburgh: E. and S. Livingstone Limited. 1953.

Contents: 1. The Law of Southern Africa and its Administration. 2. Registration and Professional Discipline in the Union and South West Africa. 3. Registration and Professional Discipline in Southern Rhodesia. 4. Judicial Powers of Supervision over the Medical Councils. 5. Unprofessional Conduct. 6. Contracts and the Practitioner: Medical Insurance. 7. Delicts and the Practitioner. 8. Crimes and the Practitioner. 9. The Public Duties of Medical Practitioners. 10. Evidence and the Expert Witness. 11. Identity. 12. The Examination of Blood Stains and the Individuality of the Blood. 13. The Diagnosis and the Early Signs of Death. 14. Necropsy Technique. 15. Deaths from Rapid Anoxia. 16. Deaths Usually Initiated by Anoxic Anoxia. 17. Deaths Initiated by Anaemic and Histotoxic Anoxia. 18. Anaesthetic Deaths. 19. Deaths from Burns, Exposure to Low and High Environmental Temperatures, and Electrocution. 20. The Medical Investigation of the Cause of Death. 21. Deaths from Acute Neurogenic Cadiovascular Failure. 22. Suspected Poisoning: The Post-Mortem Detection of Poisons. 23. Wounds. 24. Regional Injuries of Medico-Legal Importance. 25. Firearm Wounds. 26. Sexual Offences. 27. Abortion. 28. Infanticide and Concealment of Birth. 29. Medico-Legal Aspects of Acute Alcoholic Intoxication. 30. Medico-Legal Aspects of Mental Defect or Disorder. 31. Medico-Legal Aspects of Workmen's Compensation in the Union of South Africa. 32. The Law Relating to Poisons and Habit-Forming Drugs in the Union and South West Africa. 33. The Law Relating to Poisons and Dangerous Drugs in Southern Rhodesia.

There is no other book which covers the field of forensic medicine as completely as this. It is a misnomer to call it a third edition, for the previous edition has been expanded beyond recognition. Dr. Rhodes (now deceased) no longer appears as a co-author, and Dr. Price, who is responsible for the legal section, completes the trio. There are also several additional contributors, each an expert in his own field.

The book falls naturally into 2 sections, one dealing with legal medicine—about one-third of the book—and the other with forensic medicine.

The viewpoint of the medical authors is not simply that of morbid anatomists but also of physiologists and clinicians. This makes for a stimulating approach. The chapters on the interpretation of the causes of death, particularly that on acute neurogenic cardio-vascular failure, illustrate this.

The section on the examination of blood stains and the individuality of the blood is comprehensive, with emphasis on blood grouping. There is a minor defect in Fig. 26, which does not reproduce accurately the relative intensities of the absorption spectra bands; the α band should be denser than the β band in haemochromogen.

One of the best sections is that dealing with anoxia. All the syndromes producing anoxia are logically classified, and the traditional concept of death as being caused by coma, syncope and asphyxia has been discarded. The presentation adopted gives a rational basis to the causes of death and allows for correlation in a subject which, in other text-books, is dealt with in watertight compartments.

Among regional injuries of medico-legal importance, injuries of the brain are analysed on the basis of the physical forces which produced them. This gives a clear explanation of the mechanism of their production.

For the lawyer there is a wealth of material to aid cross-examination. The section on alcohol will be quoted daily in the courts. Much guidance will also be afforded by the chapters on wounds and injuries. The medical witness is often asked his opinion on the force used in delivering a blow. This section destroys the myth that one can estimate the force of a blow from mere inspection of the wound.

The chapter on the diagnosis and early signs of death is of value. Every forensic pathologist sooner or later has to give evidence on the post-mortem interval. He will find all the factors by which this is estimated lucidly set out. There is also an interesting section on the chemical basis of rigor mortis.

Toxicology has been wisely excluded, as not being within the range of the forensic pathologist.

The authors have culled world literature to bring their work up to date. In a short review one cannot deal with the excellence of the remaining chapters. As a book on forensic medicine, it is a first-class production.

In the section dealing with the law the authors have shown boldness. They have dealt with every aspect of the law which affects medical practitioners. However, the authors have cast their net too wide. To find a section on the formalities of contract and one on necropsy technique in the same work startles the reviewer. Medical law, public health and forensic medicine do not lie easily in the same pages. The ambition of the authors to include all law as it affects doctors in a book on forensic medicine has not had a happy result. What would have been ideal as 2 separate books, is incongruous as one. The only link between the medical and the legal sections is that doctors practise medicine and the law also affects doctors.

Doctors require a section setting out simply their legal responsibilities, and more detailed chapters dealing with criminal law, delict, and the public duties of doctors as being those branches of the law which affect them particularly. Instead of this they are offered commentary, discussion and lengthy extracts from judgments. There is too much for the doctor, while the lawyer does not need it as he has the information elsewhere. Also, the book unnecessarily repro-

duces too many forms which are easily procurable and which most doctors have in their desks.

The book, nevertheless, meets an important need. Doctors are becoming increasingly aware of their legal responsibilities, and the legal section gives them an answer to many of their problems. The section on delicts will be particularly helpful as it deals fully with numerous cases illustrating 'medical negligence'—the bugbear of a doctor's professional life. (Is it not bold to suggest that sterilization without medical justification is not a criminal act?)

The law side has involved a tremendous amount of research and compilation. All statutes of the Union and Southern Rhodesia, affecting the medical profession, are dealt with, and there is an extensive index of case law. A feature is made of covering Southern Rhodesian law. Both doctors and lawyers will find the law as it affects the medical profession easily accessible in this book.

The volume is very well produced, clearly printed, with a good index. The reproduction of illustrations is first-rate.

The work will be of infinite help to district surgeons, hospital administrators, public health officials and any doctor whose duties take him into the courts. The book will become a standard work of reference.

NEW JOURNAL OF FORENSIC MEDICINE

Journal of Forensic Medicine, Vol. 1, No. 1, July—September 1953. (Pp. 64. Annual subscription 42s.) Cape Town: Juta & Co. Limited. 1953.

Contents: 1. A New Medico-Legal Journal. 2. Blood Groups and Skin Colour. 3. Identification of Skeletal Remains. 4. Dentures and Individual Identification. 5. Sudden or Unexpected Deaths in Infancy. 6. X-Rays in Medico-Legal Investigation. 7. Sudden Death with Minimal Anatomical Findings. 8. Estimation of Stature from the Long Bones. 9. Estimation of Age from Cranial Suture Closure. Reviews of Books. Medico-Legal Notes and News.

Forensic Medicine has become a specialized branch of medical practice and there would appear to be a definite need for a scientific journal, international in scope, which is devoted exclusively to this broad speciality. The *Journal of Forensic Medicine*, which is sponsored by the Medico-Legal Society (Johannesburg) and which it is intended will be published quarterly, is an attempt to meet this need.

The quality of the articles in this first number, which is pleasingly produced and of convenient size, is uneven but there are several which make some contribution to present medico-legal knowledge. Thus, E. N. Keen (Cape Town) shows that, in the examination of skeletal remains, an error of less than 2½ inches cannot be relied upon in the estimation of statures from measurement of the long bones, whilst R. Singer (Cape Town) shows that it is an unreliable procedure to assess the age of a deceased person from the extent of closure of the vault sutures of the skull.

An important paper is that of K. M. Bowden (Australia), who confirms the finding of Werne and Garrow (1947) that, when infants are found dead under circumstances suggestive of mechanical suffocation, thorough post-mortem investigation will prove many of these deaths to be due to natural causes.

M. Shapiro (Johannesburg) describes the racial distribution of many of the erythrocytic antigens and concludes that the racial origin of an individual person cannot be determined from a study of these antigens, whilst L. Adelson (U.S.A.) discusses certain neurological mechanisms which may cause sudden death and decides that the post-mortem diagnosis of cardiac inhibition must be made by a process of exclusion.

To publish a journal of this very specialized nature in South Africa, which is remote from the great medical centres of the world, is an ambitious undertaking but this first number shows distinct promise and it is to be hoped that this journal, which should appeal to all practitioners who are specially interested in the science of forensic medicine, will meet with sustained success.

* * * *

The *Journal of Forensic Medicine* makes a welcome début in the field of South African medical literature. It appears quarterly under the editorship of Dr. H. A. Shapiro. It aims at providing an international forum for papers on forensic medicine and in the first issue it has succeeded admirably. There appear articles from authors in South Africa, Australia and the United States.

An excellent article on *Blood Groups and Skin Colour* by Dr. M. Shapiro (Johannesburg) contains a careful analysis of the gene-frequency distributions of blood groups in various races. The effect of miscegenation on Rh gene-frequencies in the different groups of the South African population makes the author pose the question—'which of us can claim to be of pure race?' His conclusion is that there is no pure race on earth.

Another good article is by Dr. E. N. Keen of the Anatomy Department of the University of Cape Town. He deals with the estimation of stature from long bones. He doubts the worth of the well-known Manouvrier's tables and also that of Pearson's formula. Dr. Keen is critical of the inferences drawn in the celebrated Wolkersdorfer case in South Africa.

Dr. R. Singer of the same department discusses the estimation of age from cranial suture closure and decides it is valueless.

Dr. Prinsloo (Government Pathologist, Durban) in a concise and interesting article deals with the identification from skeletal remains, as done in the Howick Falls murder in 1952.

In an article on sudden deaths in infancy Dr. Bowden (University of Melbourne), analyses the morbid pathology found in 320 cases. He draws the important conclusion that accidental suffocation is seldom the cause of unexpected death in infancy, though the superficial assessment of the circumstances might suggest this to be the case.

The remaining articles deal with X-rays as an aid to medico-legal investigations and the neurological mechanism of sudden death.

The *Journal of Forensic Medicine* will be of value to forensic pathologists, district surgeons, and all practitioners who must decide medico-legal problems. It is the only journal in international medical literature dealing with this branch and it deserves support.

OPHTHALMIC PATHOLOGY

Ophthalmic Pathology: An Atlas and Textbook. By J. S. Friedenwald, H. C. Wilder, A. E. Maumenee, T. E. Sanders, J. E. L. Keyes, M. J. Hogan, W. C. and E. U. Owens, with the editorial assistance of H. K. Steward. (Pp. 489 + ix, with 240 plates. South African price: £7 13s.) Published under the Joint Sponsorship of the American Academy of Ophthalmology and Otolaryngology and the Armed Forces Institute of Pathology. Philadelphia and London: W. B. Saunders Company. South African representatives: P. B. Mayer, Cape Town. 1952.

Contents: 1. Introduction: Anatomic and Physiologic Considerations. 2. Histology. 3. Growth and Aging. 4. Nature and Mechanism of Inflammation. 5. Endophthalmitis and Phthisis Bulbi. 6. Focal Lesions in Endogenous Endophthalmitis. 7. Granulomatous Inflammations. 8. Injuries. 9. Extrabulbar Diseases. 10. Diseases of Conjunctiva and Cornea. 11. Diseases of the Lens. 12. Intraocular Fluid Circulation, Glaucoma and Hypotony. 13. Diseases of the Ocular Blood Vessels. 14. Retina, Optic Disc and Optic Nerves. 15. Congenital and Developmental Anomalies. 16. Prenatal and Neonatal Diseases. 17. Heredofamilial and Degenerative Diseases. 18. Tumours. Index.

Ophthalmologists the world over are greatly indebted to the Armed Forces Institute of Pathology, who have combined with the American Academy of Ophthalmology and Otolaryngology in commissioning a group of distinguished contributors to prepare this ambitious publication, which is based on J. S. Friedenwald's well-known *Pathology of the Eye*. The volume combines an atlas with a text-book of unusually high standard.

It is thoroughly up to date and its value is enhanced by the opening chapter, which discusses the anatomical and physiological mechanisms that influence pathological changes. Subsequent chapters deal systematically with the whole of ocular pathology in a lucid manner and it is difficult to single out any particular section because of the excellence of the whole.

The arrangement whereby the photo-micrographs are presented together to illustrate subjects corresponding to chapter headings, rather than to specific cases, adds materially to its usefulness. These photo-micrographs, of which there are 240, are superb and the result is a pathological treatise which is beyond praise and a credit to everyone concerned. This is a book that should definitely be in the possession of every practising ophthalmologist.

The Medical Association of South Africa : Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP-AFDELING

KAAPSTAD : CAPE TOWN

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PRAKTYKE TE KOOP : PRACTICES FOR SALE

(1295) Karoo hospitaaldorp. Geleë in vooruitstrewende skaapdistrik. Ontvangste vir 1952: £2,640. Premie verlang: £900. £500 kontant, balans oor 2½ jaar. Drie aanstellings, aan die praktyk verbonde.

(1349) Eastern Province hospital town. Partnership share in large busy practice. Gross income for the last year was over £5,000. Premium required £1,250. Excellent opportunity for Afrikaans doctor interested in surgery.

(746) Cape Town. Large dispensing practice, mainly non-European. Average annual cash receipts approx. £5,200. £5,500 required for premium, drugs and surgery furniture.

(1356) Very well established CAPE TOWN SUBURBAN PRACTICE. Outright sale or alternatively partnership share available to Gentile purchaser. Excellent opportunity to acquire a good class practice. Details on application.

(1387) Boland. Nucleus praktyk en goeie voorraad instrumente, ens. teen £400. Uitstekende vooruitsigte vir uitbreiding.

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ASSISTENTE/PLAASVERVANGERS VERLANG
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(1347) Cape Town suburb. Gentile assistant with view to partnership. Salary offered £80—£100 per month according to qualifications. Locum must have own car.

(1409) Ship's surgeon from mid-March 1954 for 1 month for voyage from Cape Town to Beira and back. Male essential. Salary to be arranged.

(1410) Northern Cape. Locum tenens in partnership of three from early November for 12–15 months. Salary £75 p.m. plus £10 p.m. car allowance and free board and lodging. Own car essential. Scope for surgery.

(1414) Transkei. Locum for the period 23 September—7 November. Salary £2 12s. 6d. Own car not required.

(1426) S.W.A. Locum for 6 weeks, any time from now to 7 October. General practice. Very little night work, maternity and travelling. Salary £3 10s. per day plus board and lodging. 9d. per mile travelling expenses paid.

(1444) Noord Kaapland. Assistent dadelik benodig met oog tot vennootskap. £75 p.m. plus kartoelae.

(1443) Eastern Province. Locum from ± 14 December to ± 31 December. Salary £2 12s. 6d. per day plus board and lodging and car allowance. Preferably man.

(1438) Boland. Locum from ± 15 December 1953 for 1 year. Later possibility of assistant- or partnership. Salary offered £3 3s. per day. Preferably own car. Partnership practice.

* * * *

JOHANNESBURG

Medical House, 5 Esselen Street, Telephone 44-9134-5, 44-0817
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(L/V413) Assistant required for country practice near Johannesburg. Hospital facilities. Definite view to partnership. Preferably Gentile doctor, capable of doing surgery.

(L/419) Reef hospital town. Assistant required as from 1 September. Own car necessary. Preferably experienced man.

(L/V420) Locum for the month of October. Must have own car. Salary £2 12s. 6d. per day, free petrol and oil and board and lodging and £10 p.m. car allowance. Will suit newly qualified man.

(L/V421) An assistant is required for a very large European practice in Johannesburg, mainly Afrikaans-speaking patients. Will suit a newly qualified man. Excellent prospects.

(L/V423) O.V.S. Plaasvervanger benodig vir tydperk 4–6 maande, weens siekte van vennoot. Moet Afrikaans-sprekend wees, eie kar gebruik en verkieslik ongetroud wees. Salaris £2 2s. per dag, vry losies en petrol en olie. 6d. per myl vir ritte verder as 3 myl.

(L/V425) O.F.S. Locum required for September. Salary £3 3s. per day, and all found. Own car necessary.

PARTNERSHIP OFFERED

(P/O21) Half-share in essentially English-speaking private practice in Johannesburg. Preferably Gentile with 3 or 4 years' experience. Premium £2,500.

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PRAKTYKE TE KOOP : PRACTICES FOR SALE

(Pr/S81) Oos-Vrystaat. Geen opposisie. D.G. aanstelling teen £425 p.j. Jaarlikste inkomste £2,500. Premie van £750 sluit praktyk-toerusting, instrumente en medisyne in. As volg betaalbaar: £300 kontant en balans op maandelikse paaiemente; die bedrag waarvan onderling gereel kan word.

(Pr/S82) Excellent non-European practice near Johannesburg. Established in 1944. Average annual *net* income £2,700 cash. Premium required is £2,000 and terms can be arranged. Premium includes contents of surgery and maternity ward.

(Pr/S78) Oud-gevestigde Vrystaatse praktyk met D.G. aanstelling. Gemiddelde jaarlikse inkomste oorskrei £4,000. Premie van £2,000, sluit medisyne en apparate in. Uitstekende geleentheid vir 'n jong man.

(Pr/S84) Pleasant town in Northern Transvaal, with hospital facilities. General practice which was run by seller for 10 years besides a large non-transferable mine appointment. The appointment did not allow time for any Native work—only for very few district calls. Net cash income over £1,200 per year though only few hours daily were spent in this practice. Premium £500 on terms. Excellent start for young man.

(Pr/S85) Progressive Transvaal dispensing practice. Excellent surgical facilities. Average gross income £3,500 per annum. Premium required £2,500 and the following terms could be arranged: £1,250 deposit and the balance over a period of 18 months, starting 3 months after cash payment. The premium includes drugs, furniture and fittings, estimated at £800. Two transferable appointments worth £230 per annum. Scope for expansion.

(Pr/S87) Wes-Transvaal. Uitstekende praktyk. Gemiddelde jaarlikse inkomste oorskrei £3,000. Woonhuis en spreekkamers te koop of te huur teen £14 en £11 per maand, onderskeidelik. Premie verlang is £1,500 en terme kan gereël word. Skryf om volle besonderhede.

(Pr/S88) O.V.S. Algemene praktyk met D.G. aanstelling. Geen opposisie. Jaarlikse inkomste ongeveer £3,500. Premie van £1,750 sluit in groot voorraad medisyne, instrumente en meubels. Hierdie is ook 'n ougevestigde praktyk.

* * * *

DURBAN

112 Medical Centre, Field Street. Telephone 2-4049

PRACTICES FOR SALE : PRAKTYKE TE KOOP

(PD13) Natal Lower South Coast practice, near Pondoland border, suitable for retired doctor. Area developing and large Police holiday camp in vicinity. Excellent climate and very good fishing. Premium required £400, includes good stock of drugs and dressings, instruments and dispensary furniture. House for sale £1,800, including stand of one-third morgen. Bond available. For immediate sale. Owner having taken a full-time appointment.

(PD15) General practice established 1941 at pleasant residential and seaside resort about 10 miles south of Durban. Annual income approximately £1,000. No major surgery, minimum of minor surgery and only emergency midwifery being done at present. Brick house with consulting room attached, for sale at £5,250. Owing to ill health owner wishes to retire from practice as soon as possible. Premium £1,000 including drugs, surgery and dispensary furniture.

(PD20) Natal South Coast. General mixed prescribing practice.

Premium £1,000 plus £200 for full equipment of 2 surgeries. Large proportion of the patients are European visitors, and Indians. A lucrative Native practice could be built up if dispensing was carried out. Immediate introduction.

(PD21) East Griqualand. General mixed practice with net profit of £3,000 annually. Premium £1,900, terms if required. Excellent opportunity for newly qualified practitioner.

(PD22) Natal. Prescribing and dispensing country practice. Total gross receipts for 1951, £3,344 15s. 9d.; 1952, £2,817 10s. 6d.; 1953 (3 months), £846 6s. 10d. Premium £1,500, includes drugs, consulting room furniture and instruments. House for sale £5,500.

(PD23) Natal. Prescribing practice particularly suitable for a woman doctor interested in obstetrics and gynaecology. Total gross receipts for 1950, £1,570; 1951, £1,595; 1952, (6 months), £1,340; 1953 (3 months), £382. Premium £1,250, includes furniture, fittings, instruments, drugs and existing book debts.

PARTNER REQUIRED

(PDX) General Practitioner in Durban offers partnership preferably to one with experience. Capital necessary.

ASSISTENTE/PLAASVERVANGERS VERLANG
ASSISTANTS LOCUMS REQUIRED

(138) Assistant required immediately in general country practice near Pietermaritzburg. £1,000 per annum. Two appointments. Very little surgery or midwifery. Should possess own car.

(139) Locum required Natal country practice. 30 August to 30 September. Must be bilingual and possess own car. £2 12s. 6d. per day, all found.

(140) Assistant immediately until end of year. Partnership of four. Experience in anaesthetics a recommendation. Hospital facilities available. Salary £100 per month.

Transvaal Provincial Administration

VACANCIES: TRANSVAAL PUBLIC HOSPITALS

Applications are invited from suitably qualified candidates for the undermentioned posts at Public Hospitals in the Transvaal.

Applications should be addressed to the Medical Superintendents of the undermentioned Hospitals concerned and should contain full particulars as to the age, professional and academic and language qualifications, experience and conjugal status of the applicant and should further indicate the earliest date upon which duties can be assumed. Copies, only, of recent testimonials to be attached.

Cost-of-living allowance payable at present to full-time employees:

	Cost-of-Living Allowance	
Salary	*Married*	*Single*
Over £350 per annum	£320 per annum	£100 per annum

Full-time employees receive in addition to their salaries and cost-of-living allowance, the following privileges:

Leave and rail concession.

Successful candidates will be required to submit satisfactory certificates as also to submit to a medical examination at the hospital concerned.

Application forms are obtainable from any Transvaal Provincial Hospital or the Provincial Secretary, Hospital Services Branch, P.O. Box 2060, Pretoria.

The closing date of applications for undermentioned posts will be 14 September 1953.

Hospital	*Post*	*Emoluments*	*Remarks*
Baragwanath, Johannesburg	Deputy Superintendent (1)	£1,200×50–1,500	Registered medical practitioner. Administrative experience and qualifications a recommendation. Plus £180 per annum a house allowance.
Johannesburg Hospital Board and the University of the Witwatersrand.	Part-time 2nd Assistant Ophthalmologist (1)	£100 p.a.	Registered medical practitioner. One session per week.

(42146)

Provincial Administration of the Cape of Good Hope/University of Cape Town:

JOINT MEDICAL STAFF FOR GROOTE SCHUUR AND OTHER TEACHING HOSPITALS: VACANCIES

1. Applications are invited from registered medical practitioners (registered specialists) for appointment to the following posts:

Department of Ear, Nose and Throat—1 post of medical practitioner, Grade F—Salary £164 per annum per session (2 sessions).

Department of Obstetrics and Gynaecology—1 post of medical practitioner, Grade F—Salary £164 per annum per session (2 sessions).

2. The conditions of service are prescribed in terms of Hospital Board Service Ordinance No. 19 of 1941, as amended, and the regulations framed thereunder.

3. The Joint Medical Staff is required to serve jointly the Provincial Administration of the Cape of Good Hope and the University of Cape Town.

4. Candidates are required to have not less than 3 years' experience after registration as a specialist in the speciality in which the vacancy exists.

5. A session shall be 4 hours per week not necessarily continuous clinical and/or teaching work.

6. Applications must be made on the prescribed form, Staff 23, which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Medical Superintendent of any provincial hospital or Secretary of any School Board in the Cape Province.

7. The completed application forms must be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 30 September 1953.

A562720

Provinsiale Administrasie van die Kaap die Goeie Hoop/Universiteit van Kaapstad:

GESAMENTLIKE MEDIESE PERSONEEL VIR GROOTE SCHUUR EN ANDER OPLEIDINGS-HOSPITALE: VAKATURES

1. Aansoeke word ingewag van geregistreerde geneeshere (geregistreerde spesialiste) vir aanstelling tot die volgende poste:

Departement van Oor, Neus en Keel—1 pos van geneesheer, Graad F—Salaris £164 per jaar per sessie (2 sessies).

Departement van Vrouesiektes en Verloskunde—1 pos van geneesheer, Graad F—Salaris £164 per jaar per sessie (2 sessies).

2. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.

3. Die Gesamentlike Mediese Personeel word vereis om die Provinsiale Administrasie van die Kaap die Goeie Hoop en die Universiteit van Kaapstad gesamentlik te dien.

4. Kandidate moet minstens 3 jaar ondervinding na registrasie as 'n spesialis in die spesialiteit waarin die vakature bestaan, opgedoen het.

5. 'n Sessie is 4 uur per week in verband met kliniese en/of opleidingswerk maar is nie noodwendig onafgebroke nie.

6. Aansoeke moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Mediese Superintendent van enige provinsiale hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.

7. Die ingevulde aansoekvorms moet aan die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, gerig word en moet hom uiters op 30 September 1953 bereik. Kandidate moet die vroegste datum meld waarop hulle diens kan aanvaar.

A562720

Rhodesia Railways

VACANCIES FOR MEDICAL OFFICERS

Applications are invited from registered medical practitioners for the posts of Railway Medical Officer at Bulawayo, Salisbury, Umtali, Broken Hill and Livingstone. These vacancies are all created by expansion of staff.

Salary: £1,400 to £2,200 per annum (pensionable) at Bulawayo, Salisbury, Umtali and Broken Hill. Private practice *not* allowed at these centres. £1,065 to £1,245 per annum (pensionable) at Livingstone, where private practice will be permitted. Annual increments of not less than £50 per annum, subject to efficiency barriers, and a 3 year probationary period which carries the commencing salary of £1,400 p.a. and £1,065 p.a. respectively. Variable cost-of-living (at present 20%), and children's allowances payable.

Leave: 40 days vacation leave per annum (accumulative) plus 30 days long service leave for each completed period of 4 years' continuous service.

Experience: Previous hospital experience, general practice and anaesthetics essential.

Duties: Duties are chiefly those of a general practitioner, but at Bulawayo and Salisbury, they do not include the attendance on hospitalized patients. At all centres they include conducting an African Clinic and other duties as allocated by the Chief Medical Officer.

Housing: Unfurnished house provided at a rental of approximately £10 per month, except at Livingstone.

Further information and particulars will be supplied to suitable applicants.

Applications, accompanied by copies of recent testimonials, stating age, qualifications, previous experience, marital state, nationality, birthplace and name of 2 persons to whom reference can be made, should be forwarded to The Chief Medical Officer, Rhodesia Railways, P.O. Box 792, Bulawayo. (MD123)

Public Service Vacancies

1. The attention of medical practitioners, registered with the South African Medical and Dental Council, is drawn to an advertisement appearing in the *Government* and *Provincial Gazettes* of 21 and 28 August and 4 September 1953, inviting applications for the undermentioned posts:

Posts	*Department*	*Salary Scale*
Medical Inspector of Hospitals	Cape Provincial Administration	£1,300 × 50–1,500
Medical Inspector	Health (Durban, Bloemfontein and Johannesburg)	£1,000 × 50–1,200
Medical Officer	Health (Nelspoort Sanatorium and Kimberley)	£900 × 50–1,150
Medical Officer (Silicosis Medical Bureau)	Mines (Johannesburg)	£900 × 50–1,050 or £1,000 × 50–1,200
Medical Officer (on contract for two years)	Health (White River, Stellenbosch, Bethlehem and Cradock)	£900 × 50–1,150

2. In addition to salary a cost-of-living allowance at the rate of £320 per annum (married) and £100 per annum (single) is payable at present.

3. It is emphasised that full and detailed particulars of qualifications and previous experience must be furnished but original certificates and testimonials should not be submitted. Application forms Z.83 and P.S.C. 8(*a*) are obtainable from the Secretary for Health, Pretoria, the Secretary for Mines, Pretoria, or the Provincial Secretary, Cape Town, as the case may be, and filled in forms must be returned to them.

4. The closing date for the receipt of applications is 26 September 1953. (42037)

Nasionale Hospitaal: Bloemfontein

Aansoeke word hiermee ingewag van kandidate met geskikte kwalifikasies vir die volgende pos by die Nasionale hospitaal en Tempe Provinsiale Hospitaal, Bloemfontein.

Aansoeke moet gerig word om die Geneesheer-Direkteur so spoedig moontlik te bereik en moet volle besonderhede bevat aangaande die ouderdom, professionele kwalifikasies, ondervinding en huwelikstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop diens aanvaar kan word indien aangestel.

(a) Voltydse Narkotiseur-spesialiteit op die salarisskaal £1,750 × 50—£1,900 p.j. plus heersende lewenskostetoelae, tans £320 p.j. vir getroude persone en £100 p.j. vir ongetroude persone.

Van die suksesvolle applikant sal verwag word om bevredigende sertifikate in te dien aangaande kwalifikasies.

Alle aanstellings geskied in terme van die Hospitaal Regulasies soos gewysig.

J. W. Wessels
Geneesheer-Direkteur

18 Augustus 1953 (A375533)

Assistant or Locum Wanted

Large mixed practice, mainly non-European. Major Rhodesian city. Male or female, but single person preferred. Salary to be based on experience. Duties to commence on 1 December 1953. Write to 'A. R. Y.', P.O. Box 643, Cape Town.

Assistantship Required

Experienced general practitioner (gentile) requires assistantship with view, in Cape Province or Natal. Capital available. Write 'A. R. W.', P.O. Box 643, Cape Town.

Printed by Cape Times Ltd., Parow, and Published by the Proprietors, THE MEDICAL ASSOCIATION OF SOUTH AFRICA, MEDICAL HOUSE, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'